A Dissertation on

"A STUDY TO DETERMINE THE PREVALENCE OF PRIMARY OPEN ANGLE GLAUCOMA IN PATIENTS WITH SYSTEMIC HYPERTENSION"

Submitted to the

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements

For the award of degree of

M.S. (Branch – III)

OPHTHALMOLOGY

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL

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APRIL 2014

CERTIFICATE

This is to certify that the study entitled **"A STUDY TO DETERMINE THE PREVALENCE OF PRIMARY OPEN ANGLE GLAUCOMA IN PATIENTS WITH SYSTEMIC HYPERTENSION"** is the result of original work carried out by **Dr. INDHU. C,** under my supervision and guidance at **STANLEY MEDICAL COLLEGE, CHENNAI.** The thesis is submitted by the candidate in partial fulfilment of the requirements for the award of M.S Degree in Ophthalmology, course from May 2011 to April 2014 at Stanley Medical College, Chennai.

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DECLARATION

I hereby declare that this dissertation entitled **"A STUDY TO DETERMINE THE PREVALENCE OF PRIMARY OPEN ANGLE GLAUCOMA IN PATIENTS WITH SYSTEMIC HYPERTENSION"** is a bonafide and genuine research work carried out by me under the guidance of **Prof. Dr. K.KANMANI M.S.,D.O.,** Professor, Department of Ophthlamology, Government Stanley Medical College and Hospital, Chennai- 600001.

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ABSTRACT

“A STUDY TO DETERMINE THE PREVALENCE OF PRIMARY OPEN ANGLE GLAUCOMA IN PATIENTS WITH SYSTEMIC HYPERTENSION"

Key Words Primary Open Angle Glaucoma, Intraocular Pressure, Systemic Hypertension

OBJECTIVES OF THE STUDY:

The aim of the study was to find the prevalence of primary open angle glaucoma in patients with systemic hypertension presenting to Stanley OPD to find the association of systemic hypertension with the occurrence of POAG.

METHODS:

In this cross-sectional case control study, 100 patients with systemic hypertension and 100 non hypertensive subjects were evaluated

at department of ophthalmology at Stanley medical college for the presence of primary open angle glaucoma based on predetermined criteria and the prevalence of primary open angle glaucoma in both the groups was found and analysed to find the asscociation of primary open angle glaucoma and systemic hypertension.

RESULTS:

Systemic hypertension was not found to have a statistically significant association with the occurrence of primary open angle glaucoma{ odds ratio 1.936 with 95% confidence interval being from 0.687 to 5.457. The mean intraocular pressure of hypertensive group was mildly higher than the mean intraocular pressure non hypertensive group and the difference in the mean IOP of both the groups was statistically significant { P value – 0.006}. The occurrence of POAG was more in patients with systemic hypertension for a longer duration i.e 55.5% of persons with POAG in this group was diagnosed with hypertension for ≥ 10 years. The prevalence of POAG also increased with increasing age in both the groups with 66.6% of subjects being above 60 years of age.

Conclusion:

In our study systemic hypertension was not found to have a statistically significant association with POAG,but persons with long duration of hypertension and advancing age, need to be monitored for high IOP and optic nerve head changes to aid in early diagnosis and also to minimize visual morbidity and blindness due to glaucoma.

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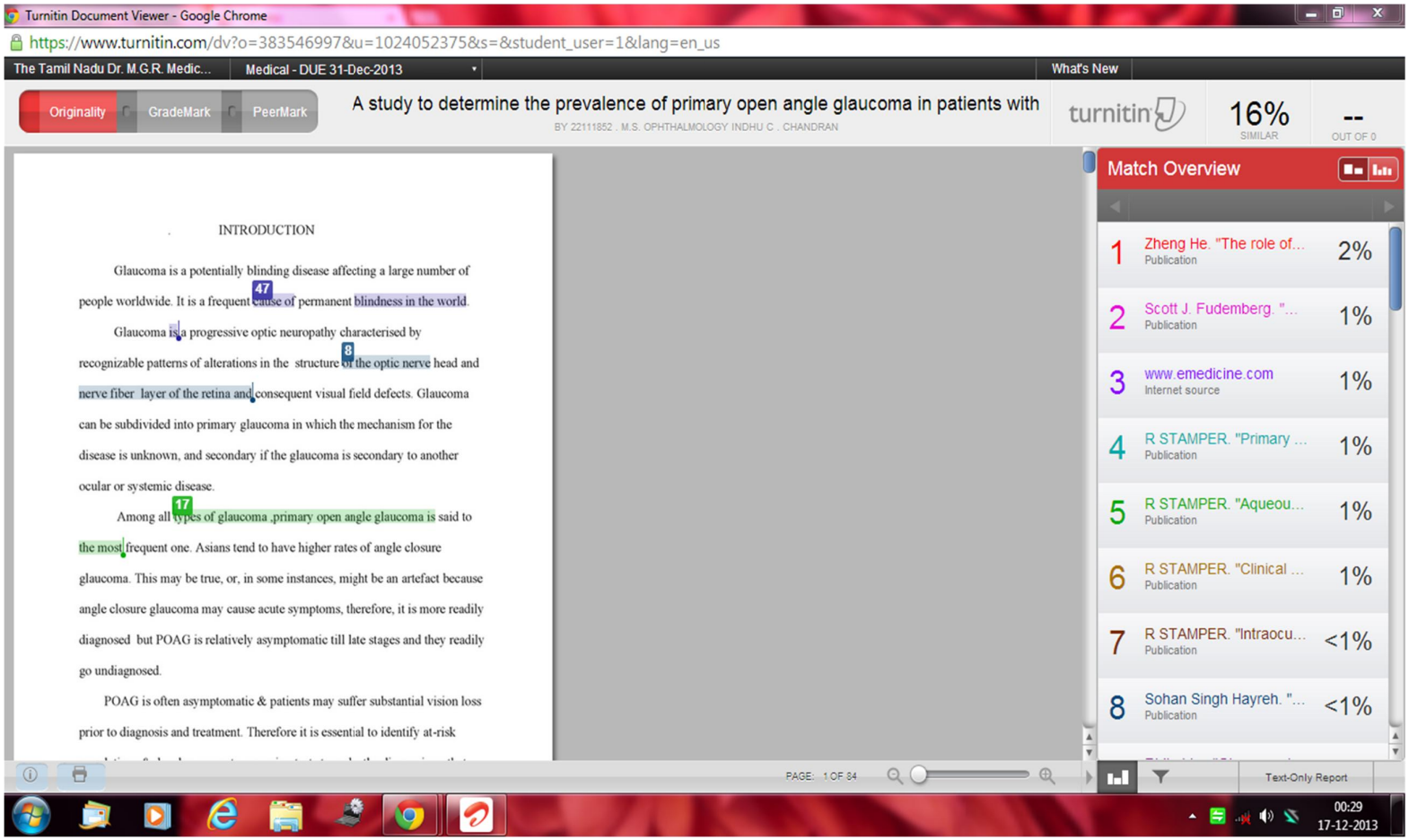
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INTRODUCTION
Glaucoma is a potentially blinding disease affecting a large number of people worldwide. It is a frequent cause of permanent blindness in the world.
Glaucoma is a progressive optic neuropathy characterised by recognizable patterns of alterations in the structure of the optic nerve head and nerve fiber layer of the retina and consequent visual field defects. Glaucoma can be subdivided into primary glaucoma in which the mechanism for the disease is unknown, and secondary if the glaucoma is secondary to another ocular or systemic disease.
Among all types of glaucoma ,primary open angle glaucoma is said to the most frequent one. Asians tend to have higher rates of angle closure glaucoma. This may be true, or, in some instances, might be an artefact because angle closure glaucoma may cause acute symptoms, therefore, it is more readily diagnosed but POAG is relatively asymptomatic till late stages and they readily go undiagnosed.
POAG is often asymptomatic & patients may suffer substantial vision loss prior to diagnosis and treatment. Therefore it is essential to identify at-risk
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INTRODUCTION

Glaucoma is a potentially blinding disease affecting a large number of people worldwide. It is a frequent cause of permanent blindness in the world.

Glaucoma is a progressive optic neuropathy characterised by recognizable patterns of alterations in the structure of the optic nerve head and nerve fiber layer of the retina and consequent visual field defects[6]. Glaucoma can be subdivided into primary glaucoma in which the mechanism for the disease is unknown and secondary if the glaucoma is secondary to another ocular or systemic disease.

Among all types of glaucoma, primary open angle glaucoma is said to the most frequent one. Asians tend to have higher rates of angle closure glaucoma. This may be true, or, in some instances, might be an artefact because angle closure glaucoma may cause acute symptoms, therefore, it is more readily diagnosed but POAG is relatively asymptomatic till late stages and they readily go undiagnosed.

POAG is often asymptomatic & patients may suffer substantial vision loss prior to diagnosis and treatment[7]. Therefore it is essential to identify at-risk populations & develop accurate screening tests to make the diagnosis so that sight-preserving treatment can begin early in the disease process. Several population based studies have been carried out to determine the prevalence of

POAG and to identify risk factors for the disease. Age[8,9,10], race[12], myopia[17], systemic HT[11,15], diabetes[11,13,14], family history[18] etc have long been considered as risks for occurrence of primary open angle glaucoma but recent studies have questioned the strength of these associations.

Blood pressure increases with age in most populations but the relationship between systemic hypertension and POAG is yet to be successfully elucidated. Several large-scale studies have yielded dissimilar & even contradictory results. Systemic HT causes direct microvascular damage and can impair blood flow to the optic nerve, precipitating POAG. Significant ethnic & racial variation exists in the prevalence and complications of HT. Evaluation of hypertension as a risk factor for POAG in our population is essential so that the disease can be detected at an early stage by screening these high risk groups.

REVIEW OF LITERATURE

HISTORIC REVIEW:

Glaucoma has been known since Roman era. The word "glaucoma" was first described in Hippocrates' *Aphorisms* about 400 BC as an ailment of old men.!

In Hippocratic writings,' *glaucosis'* derived from the Greek word *glaucos,* meaning glaze , later more specified as a greenish glaze of the lens.

In 1626 Dr.Banister first established the concept of elevated intra-ocular pressure in glaucoma.

Impairment of the aqueous drainage of the eye as the cause of glaucoma was described by Hermann Boerhave in 1708.Inflammation as a cause of glaucoma was put forth in 1722 by Charles Saint.

Emphasis was put on the intraocular pressure in the diagnosis of glaucoma till the introduction of ophthalmoscope in 1853 following which optic disc changes in glaucoma was recognised.

In 1862, Donder published his classical paper in which he described ‘Glaucoma simplex’ as a disorder with increasing tension of the eye, excavation of the disc, shift of the vessels in the disc, loss of visual field and loss of vision.

Today glaucoma is defined as a disease causing progressive atrophy of the optic nerve with loss of ganglion cells and their axons along with specific visual field defects.

ANATOMY

Aqueous humor is produced in ciliary body and drained via anterior chamber angle. These two structures play an important role in maintaining aqueous humour dynamics and also intraocular pressure.

CILIARY BODY

In the eye uveal tract is composed of iris,ciliary body and choroid. Ciliary body is seen between iris and choroid and is attached with sclera spur anteriorly. Histologically it is composed of epithelium, vascular structures and ciliary body muscle.

PARTS OF THE CILIARY BODY

- **Pars plicata:**

Pars plicata contains the ciliary processes which are the site of aqueous production, they are nothing but ridges which are radially oriented and projecting into the posterior chamber. Pars plicata forms anterior one third of ciliary body.

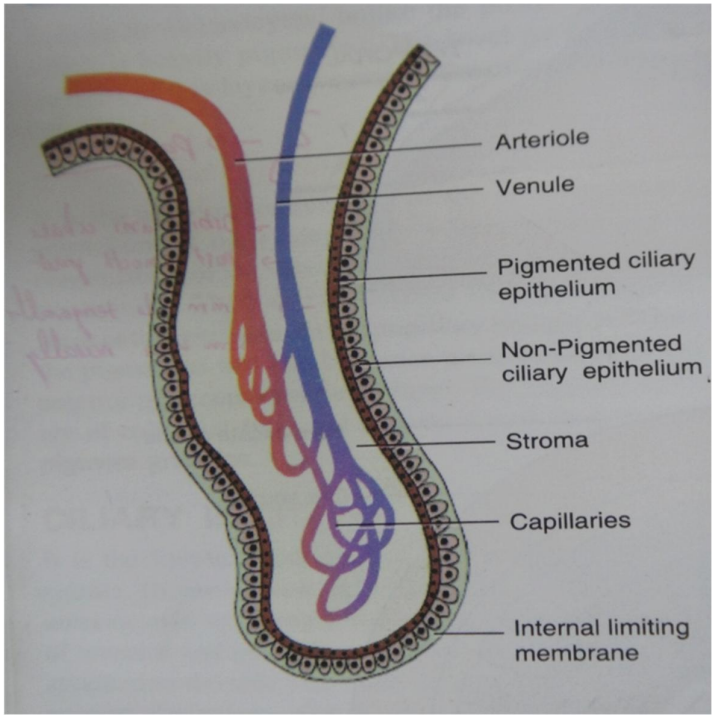
- **Pars plana:**

Pars plana is smooth, devoid of ridges and forms the posterior two third of ciliary body.

HISTOLOGY OF CILIARY PROCESS

Ciliary process is made up of a core of connective tissue substance with numerous capillaries and a overlying epithelium. Capillary network in ciliary processes are fenestrated.

Epithelium of ciliary process is double layered with an inner layer of non pigmented epithelium and outer layer of pigmented epithelium lying adjacent to the stroma. Blood aqueous barrier is formed by the tight junctions between the cells of the non pigmented epithelium.



ANGLE OF ANTERIOR CHAMBER

Schwalbe's line, trabecular meshwork, canal of Schlemn and the scleral spur are the main structures forming the anterior chamber angle.

SCHWALBE'S LINE

The anteriormost structure of the anterior chamber angle is the Schwalbe's line and it is the point where descemet's membrane ends , it also marks the point where corneal endothelium continues as trabecular endothelium.

TRABECULAR MESHWORK

The trabecular meshwork is an important structure of the anterior chamber angle playing a vital role in the maintainence of IOP, it is triangular in outline with its apex directed towards Schwalbe's line and base towards sclera spur.

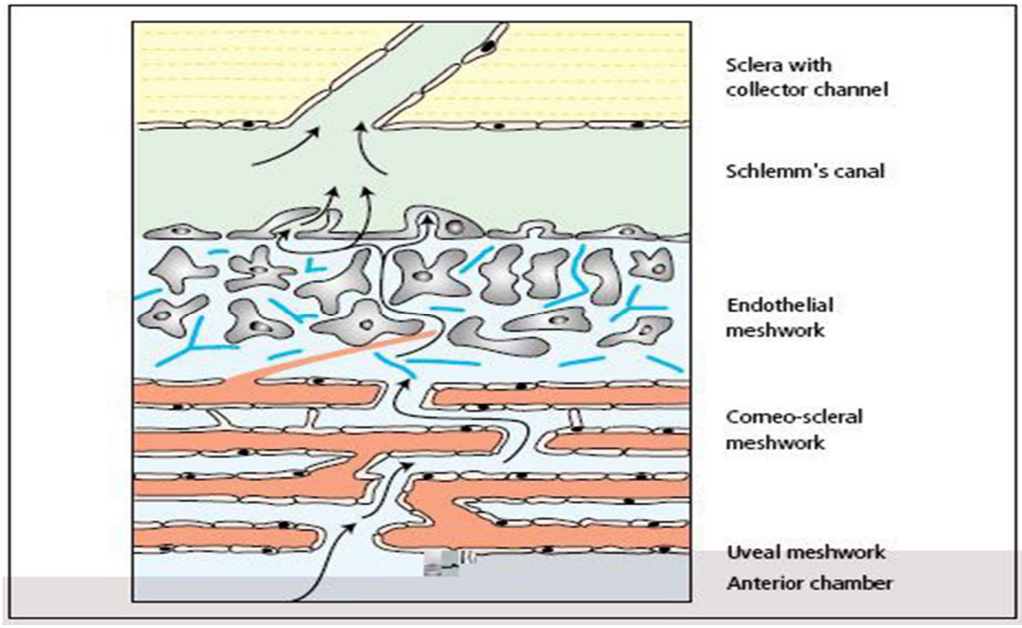
PARTS OF TRABECULAR MESHWORK

- Uveal meshwork
- Corneoscleral meshwork

■Juxta-canalicular meshwork

The inner layers of trabecular meshwork is formed by uveal meshwork, the next layer is formed by corneoscleral meshwork and it extends from anterior part of sclera sulcus to sclera spur.

Juxtacanalicular meshwork is considered as the main site of resistance for aqueous outflow and it is made up of proteoglycan matrix with embedded collagen and endothelial cells connected by gap junctions.



SCHLEMN'S CANAL

Schlemn's canal is a vascular channel lying within the sclera sulcus. It consists of two layer of endothelial cells. The outer layer is attached to sclera. The inner wall plays an important role in IOP maintenance by its invaginations called ' giant vacuoles'[6,7]. These giant vacuoles increase in size and number when IOP increases facilitating aqueous outflow thus reducing IOP.

Schlemn's canal also contains collector channels which drain aqueous into the aqueous veins which in turn drain into episceral and conjunctival veins. These collector channels are of three types namely direct (4-6) which connects directly with aqueous veins, indirect type (15-20) which forms an intrascleral plexus and then drain into aqueous veins and an intermediate type (4-6)

SCLERAL SPUR

The scleral spur is nothing but a wedge shaped projection from the anterior sclera and attaching itself anteriorly onto the trabecular meshwork. Posteriorly is attached to sclera and longitudinal ciliary muscle fibres.

As sclera spur is attached posteriorly to ciliary muscle, when ciliary muscle contracts it pulls the sclera spur posteriorly which in turn pulls the trabecular lamellae attached to it. This results in widening of intertrabecular

spaces and also prevents the schlemn’s canal to get narrowed or collapsed, in this way they help in aqueous humor outflow.

OUTFLOW OF AQUEOUS

CONVENTIONAL OUTFLOW

Outflow of aqueous from the anterior chamber angle through the trabecular meshwork into the schlemn’s canal which in turn drains into the episcleral veins via aqueous veins is considered as the conventional outflow pathway and as it drains through the schlemn’s canal it is also termed the canalicular pathway.

Contraction of the ciliary muscle through its insertion into the trabecular meshwork increases pore size in the meshwork and hence the rate of aqueous drainage. Passage of aqueous into Schlemn's canal depends on cyclic formation of transcellular channels in the endothelial lining. The collector channels from Schlemm's canal drain into the episcleral veins via aqueous veins which are about twelve in number.

UVEOSCLERAL OUTFLOW

It is estimated that up to 15% (much higher values suggested by recent studies) of aqueous outflow occurs via an alternative route known as UVEOSCLERAL OUTFLOW described in 1960s by Bill.

In this pathway aqueous enters the ciliary body course through the loose connective tissue and then pass between ciliary muscle fibres to enter supraciliary space, subsequently into suprachoroidal space. From here aqueous is drained by the veins draining the uvea. But this pathway is not affected by changes in IOP and remains the same both in high and low intraocular pressures but outflow can be modified pharmacologically. Uveoscleral outflow is decreased by pilocarpine and increased by drugs like adrenaline and prostaglandin analogs.

GRADING OF ANGLE

In an open angle structures seen from posterior to anterior are the root of the iris,ciliary body, scleral spur, trabecular meshwork and the Schwalbe's line.

Anterior chamber angle is graded by various systems namely,

SHAFFER GRADING SYSTEM

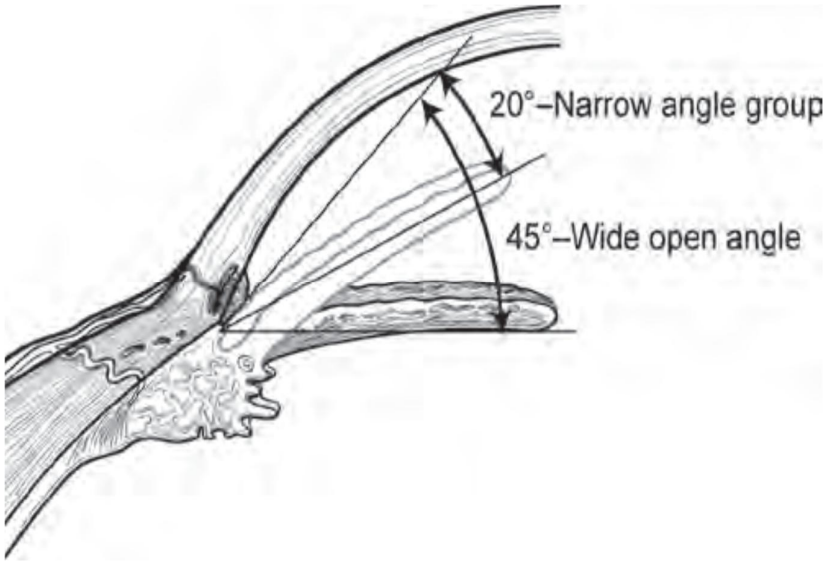
SCHEIE GRADING SYSTEM

SPAETH GRADING SYSTEM

SHAFFER'S GRADING SYSTEM

In Shaffer's grading system anterior chamber angle is graded by taking into account the angle formed between the anterior iris surface and posterior corneal wall.[25]

SHAFFER'S GRADING SYSTEM



Grade	Angle Width	Description
4	45-35	**Wide open**
3	35-20	**Wide open**
2	20	**Narrow**
1	10	**Extremely narrow**
S	<10	**Slit**
0	O	**Closed**

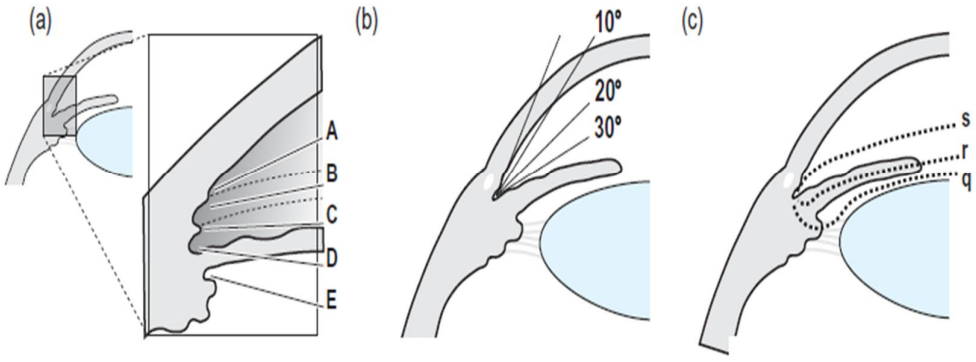
SPAETH GRADING SYSTEM

Spaeth grading of anterior chamber angle is based on three variables

a) Insertion of iris root

b) Angular width

c) Configuration of iris



Insertion of iris root

A - Anterior to Schwalbe’s line.

B - Behind Schwalbe’s line.

C - Centered at scleral spur

D - Deep to scleral spur

E - Extremely deep in ciliary body.

Configuration of iris

s - ‘Steep’ or convexly configured

r - ‘Regular’ or flat

q - ‘Queer’ for deeply concave

SCHEIE GRADING SYSTEM

This system is based on the extent of angle structures visualized

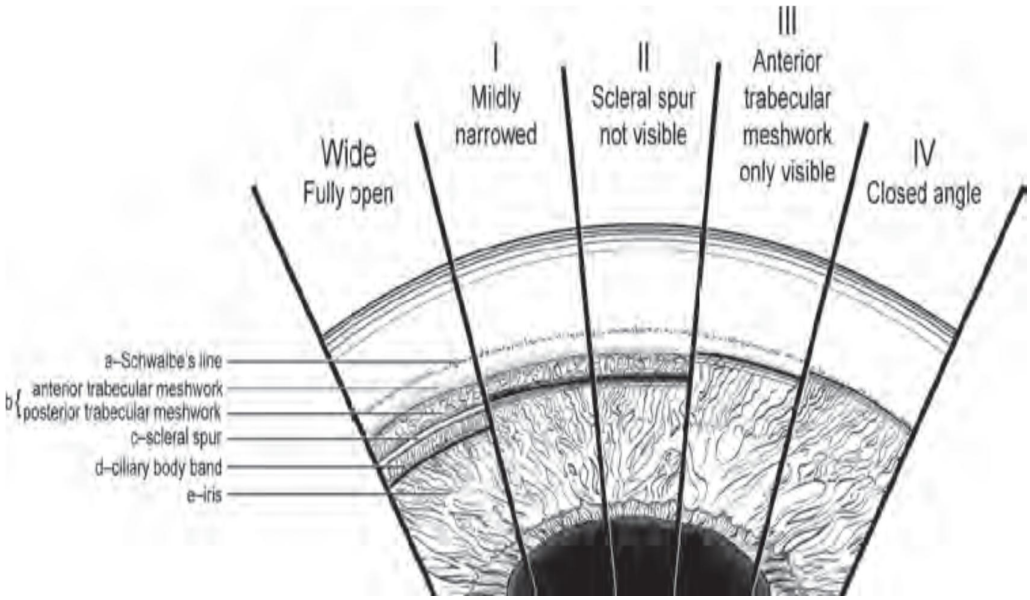
All structures seen -Wide open

Iris root not seen - Grade I narrow

Ciliary body band not seen - Grade II narrow

Posterior trabeculum obscured - Grade III narrow

Only Schwalbe’s line visible - Grade IV narrow



SCHEIE GRADING SYSTEM

GONIOSCOPY

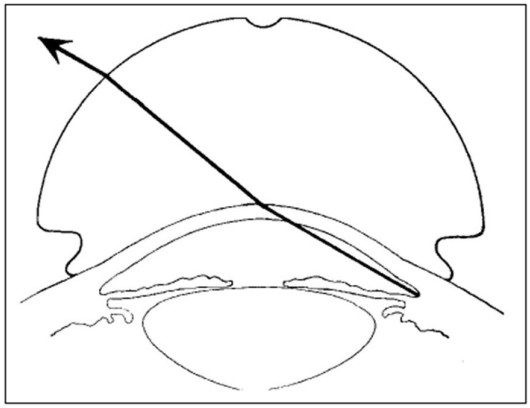
Gonioscopy refers to the technique used to view and define the structures and abnormalities of anterior chamber angle or iridocorneal angle.

PRINCIPLE

As the light emitted from angle structures undergo total internal reflection, it is not possible to visualise the angle through intact cornea. Contact lenses have an index of refraction similar to that of the cornea, allowing light to enter the lens and then be refracted (goniolens) or reflected (gonioprism) beyond the contact lens-air interface. Gonioscopy can be done by two methods i.e direct and indirect gonioscopy.

Direct gonioscopy:

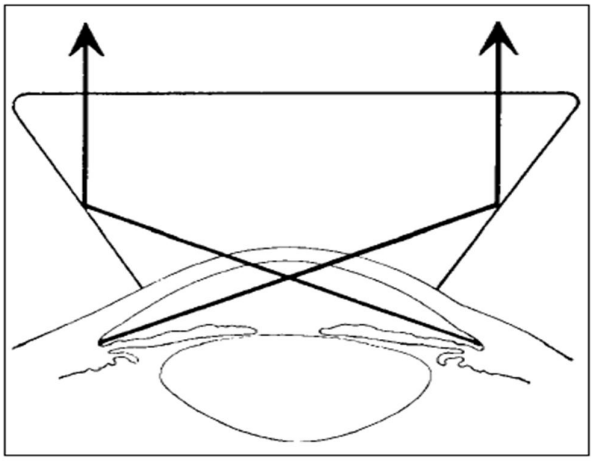
In this method,anterior curve of the contact lens i.e configuration of the contact lens is such that exiting light rays strike the contact lens/air interface at a steeper angle than critical angle so that they will pass directly to the observer without reflection inside the lens e.g is koeppe lens.



Rays of light from the angle, emerging through a Koeppe lens.

Indirect gonioscopy :

In indirect gonioscopy light rays from the angle are reflected by a mirror such that they exit the lens at an angle much less than the critical angle. So the angle viewed is the angle exactly opposite to the mirror. Indirect gonioscopy is done with the help of a slit lamp. Examples are Goldmann and Zeiss types of lenses.



Rays of light emerging through a Zeiss indirect gonioscopic lens.

AQUEOUS HUMOR:

Aqueous humor flows from its production site(nonpigmented epithelium of ciliary body) into the posterior chamber, then into the anterior chamber passing between the posterior iris surface and lens .It exits from the anterior chamber via trabecular and nontrabecular routes. The trabecular route is at the angle of the anterior chamber flowing through the trabecular meshwork (TM) of the sclera, into Schlemm's canal and then via its efferent channels,the aqueous is carried to the episcleral vessels, where aqueous mixes with blood. A balance between aqueous humor production and the resistance to its outflow is essential to maintain a normal intraocular pressure.

AQUEOUS HUMOUR DYNAMICS

It is important to understand the aqueous humour dynamics for the evaluation and management of glaucoma. Aqueous humour is formed in the ciliary process by three important mechanisms namely simple diffusion, ultrafiltration and active secretion. Active secretion of ions across the epithelium of the ciliary body is considered as the primary mechanism for the formation of aqueous these days. Active secretion creates an osmotic gradient which in turn leads to passive flow of water into the posterior chamber. This process is decreased by hypoxia, hypothermia and any inhibitor of active metabolism.

Aqueous humor exits the eye via two pathways. The trabecular meshwork is considered the conventional, pressure-dependent pathway, while the uveoscleral pathway is considered the unconventional, pressure-independent pathway.

Mean outflow facility is 0.22 to 0.33 microlitre/min/mmHg. Outflow facility decreases with age, surgery, trauma, medications, endocrine factors etc.[7]

Trabecular outflow accounts for 5 to 95% of aqueous drainage. This flow is pressure dependent, meaning that the flow is proportional to the difference between IOP and the hydrostatic pressure in the canal.

In the uveoscleral outflow pathway, aqueous humor exits the eye through the interstitial spaces between the ciliary muscle fibres into the supraciliary and suprachoroidal space where it is absorbed into the venous system. There are age-related changes to both the TM and the uveoscleral outflow pathways, including decreased TM cellularity with age and increased extracellular depositions in both the TM and uveoscleral pathways that are associated with age-dependent decreased aqueous outflow.

INTRAOCULAR PRESSURE

Although Intraocular Pressure is not part of the definition of glaucoma, reduction of IOP remains the only proven and approved means of glaucoma management and is the single most important modifiable risk factor. To cause glaucomatous optic neuropathy, there is a complex interaction between IOP and other risk factors. Therefore the study of those elements that contribute to the production and drainage of aqueous humor, maintenance of intraocular pressure and variation of intraocular pressure is material to understand the pathophysiology of this disease.

DETERMINANTS OF INTRAOCULAR PRESSURE

Intraocular pressure (IOP) is determined by three factors namely aqueous formation (F), facility of outflow (C), and episcleral venous pressure (Pv) . Goldmann equation relates these factors by the formula

$$Po = F/C + Pv ,$$

or if solving for F then

$$F = (Po - Pv) C$$

in which Po represents the IOP in the undisturbed eye in mmHg, F represents aqueous formation in ul/min, C is aqueous outflow facility in ul/min/mmHg and Pv stands for episcleral venous pressure in mmHg.

From the equation, it is clear increase in IOP occurs when the aqueous formation rate increases the episcleral venous pressure increases or the outflow facility decreases.

NORMAL INTRAOCULAR PRESSURE:

The range of intraocular pressures in the normal population is fairly wide; the average intraocular pressure is approximately 16 mmHg with a standard

deviation of 2.5. The ‘statistical’ normal range, defined as the mean two standard deviations, would therefore be approximately 11–21 mmHg.

IOP is influenced by number of factors like age, sex, race, heredity, obesity, posture, exercise etc. IOP is also altered by cholinergic and adrenergic inputs Corticosteroids raise IOP; diabetes associated with increased IOP; myopic individuals have higher IOP.

Diurnal variation -Most people have a diurnal pattern, IOP varies with an average of 3–6 mmHg in normal individuals.

TONOMETRY

Intraocular pressure is measured clinically using tonometers. Tonometers work on the principle that the pressure within the globe is directly related to the force required to deform the same.

Tonometers developed for the purpose of measuring intraocular pressure fall into two categories:

Indentation Tonometers – Here the amount of corneal or globe deformation in response to an externally applied weight is determined.

Applanation tonometers – Here the force required to flatten a surface area of cornea is determined.

In both applanation and indendation tonometers, the factors that decide the measured IOP includes the actual intraocular pressure, central corneal thickness and corneal deformability.Applanation tonometer is considered as the gold standard tool for IOP measurement.

INDENTATION TONOMETERS

Shiotz in 1905 described the indendation tonometer. The Schiotz tonometer is an indentation instrument which measures IOP by registering the depth of indentation of the cornea produced when the instrument with a known weight is applied to the eye. The weight is carried on a plunger and when the weight is applied to the eye, the intraocular pressure provides a counterbalancing force which pushes back up on the plunger. This causes a deflection of the pointer along the inclined scale. Each unit on the scale, which ranges from 1 to 20, corresponds to an indentation of 1/20 of a mm in the cornea. High intraocular pressure resists indentation, resulting in low scale readings, while low intraocular pressure allows for easy indentation, manifested by high scale readings. The intraocular pressure is determined by referring to the calibration chart and reading the pressure that corresponds to the scale reading for the plunger load applied.

Advantages:

Low cost

Portability

Disadvantages:

Ocular rigidity tends to influence the tonometry readings, example in patients with low ocular rigidity like myopia it tends to give a falsely low IOP.

APPLANATION TONOMETERS

Applanation tonometers are of two types :

1. Variable area – Here force is kept constant by using a fixed weight and the diameter of the area flattened is measured and this value gives an estimate of IOP. The prototype of this principle is Maklakov tonometer.

2. Variable force - Here area applanated is kept constant and the force required to applanate this fixed area determines the IOP. The prototype is the Goldmann applanation tonometer, which was introduced in 1954. Others include Perkins applanation tonometer, Mackay Marg tonometer, Tonopen etc.

GOLDMANN APPLANATION TONOMETER

Goldmann applanation tonometry is still considered the gold standard technique for measurement of IOP.

In using the Goldmann applanation tonometer, the flattening force to the cornea is supplied by a coiled spring contained in the instrument, controlled by a rotating knob at the base, and is applied to the anesthetized eye by the tip of a split prism device through which the cornea may be viewed with the slit lamp. The area of cornea flattened is 3.06 mm in diameter. Topical anesthetic and fluorescein are applied to the eye, and the tear film illuminated using the cobalt blue filter of the slit lamp. As the instrument is applied to the eye, the applanating head creates a circular tear film meniscus which may be viewed through the slit lamp. The prism in the applanating head splits the circular image into two semicircles, and the end point of the measurement is determined by adjusting the knob until the inside edges of the semicircles are just touching. The intraocular pressure is then determined by multiplying the reading on the scale on the knob by ten.

Source of errors of applanation tonometry:

Corneas that are too thin or thick can cause errors in IOP measurement.

Astigmatism >3 diopters can give false values. Too much of flourescein staining as well as inadequate staining can cause thick or thin mires and show inaccurate intraocular pressure.

Advantages:

Accurate method of intraocular pressure measurement

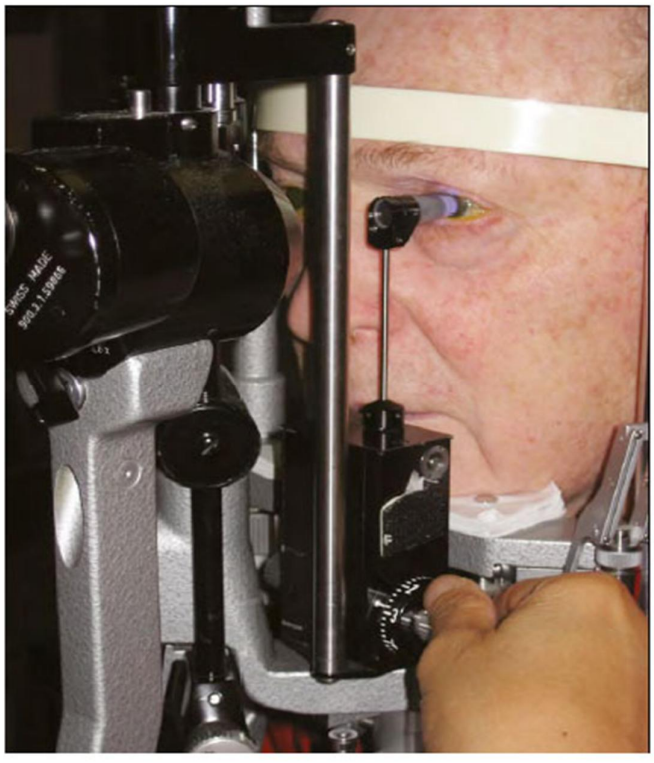
Accurate IOP measurement in patients with scleral rigidity.

Disadvantages:

Cannot be used in infants and children

Cannot be used in operative room

Goldmann tonometry will not be accurate if the cornea is very much thinner or thicker than average, highly astigmatic, irregular, edematous or scarred



NONCONTACT TONOMETERS

In non contact tonometers, cornea is not touched and flattened with a puff of air and the time required to flatten it is measured and correlated with IOP. Ocular response analyser a type of non contact tonometer is gaining popularisation in recent years.

Advantages:

No corneal trauma

No risk of infection

OPTIC NERVE HEAD ANATOMY AND BLOOD SUPPLY

Glaucoma is essentially an optic neuropathy and damage to the optic nerve is occurs within the neural, cellular and connective tissues of the optic nerve head. Knowledge about the anatomy of the normal optic nerve and the pathological changes that occur in glaucomatous optic neuropathy (GON) is essential for the early detection and monitoring of these diseases.

The optic nerve (cranial nerve II) is formed by the axons of the ganglion cells of the retina; it then traverses the scleral canal to exit the eye. Optic nerve ends at the level of optic chiasma where the axons of one side merge with axons of the contralateral optic nerve.

From its origin in the eye until it reaches the optic chiasma in the anterior cerebral fossa the ON can be divided into four segments

Intraocular part: 1 mm

Intraorbital part: 30 mm

Intracanalicular part: 6-9 mm

Intracranial part: 10 mm

The intraocular portion of the ON is also referred to as the optic nerve head (ONH). The visible most anterior part of the ONH is known as the optic disc.

Optic nerve head consists of the following zones:[7]

Surface nerve fibre layer

Prelaminar region

Lamina cribrosa region

Retrolaminar region

SURFACE NERVE FIBRE LAYER

Anteriormost part of the optic nerve head is called as the surface nerve fiber layer and it consists of approximately one million ganglion cell axons (also known as nerve fibers) from all over the retina that converge onto this part and exit through the scleral canal. Small branches from retinal arterioles and cilioretinal arteries supply the superficial nerve fibre layer.

PRELAMINAR REGION

Arterial branches from the peripapillary choroid and short posterior ciliary arteries supply the pre-laminar region.

LAMINAR REGION

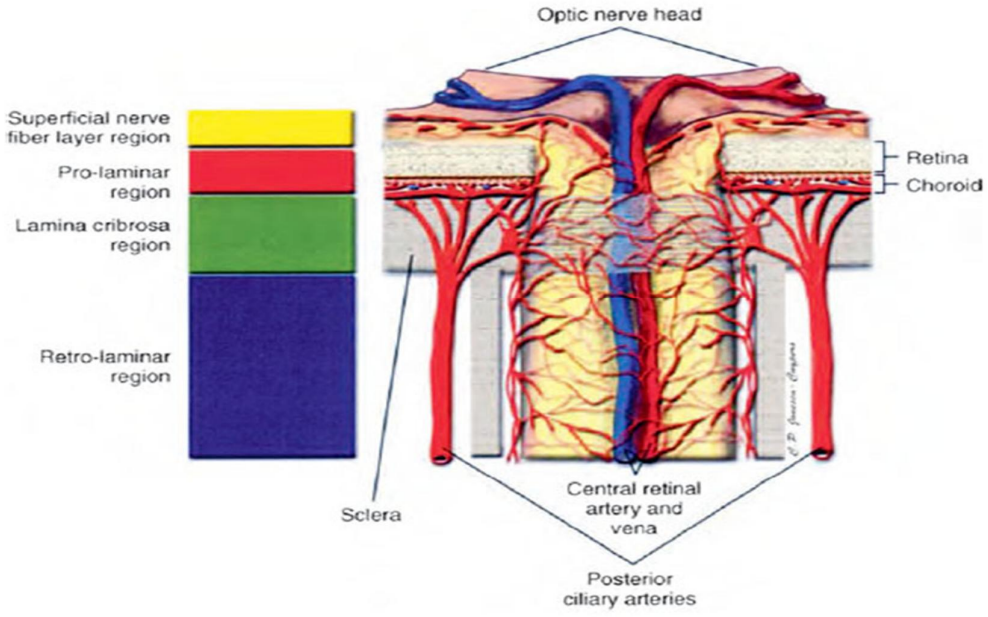
The lamina cribrosa consists of a meshwork formed by interlinked connective tissue plates (cribriform plates).Central retinal vessels and bundles of axons pass through a series of round or oval apertures within the meshwork.

The 300–400 pores that transmit axon bundles show a considerable variation in size, with the largest pores typically found in the superior and inferior quadrants. Banches of short posterior ciliary arteries either directly or its branches from circle of Zinn –Haller supplies lamina cribrosa.

RETROLAMINAR REGION

Axons within the post laminar optic nerve are myelinated, which principally accounts for the doubling of the optic nerve diameter from 1.5mm at the pre-laminar and laminar levels to 3.0 mm in the post-laminar region. Retrolaminar part supplied by centrifugal branches from central retinal artery and centripetal branches from pial vascular plexus.[30]

Venous drainage occurs via the central retinal vein and there may also be some drainage into the peripapillary choroid



OPTIC NERVE HEAD FEATURES

Upon clinical examination the optic disc is generally described as consisting of Neuraretinal rim (the nerve axons)

Optic cup(the central area of the disc surrounded by the neural rim).

CUP DISC RATIO

The optic cup is relatively devoid of nerve fascicles and normally appears as a round to oval depression of variable size, usually of a lighter color.

The diameter of the optic cup divided by the diameter of the optic disc is known as the cup/disc ratio (C/D ratio) and is expressed in decimal notation (0.1, 0.2, etc.). Ninety per cent of normal individuals have an average C/D ratio of less than 0.5 measured by direct ophthalmoscopy.

The size of the optic cup changes proportionally with the size of the optic disc; a larger cup occurs in larger disc and vice versa. Assuming discs of equal size, both optic cups usually appear fairly symmetric in normal individuals. Only 1–2% has more than a 0.2 difference between the cups.

NEURORETINAL RIM

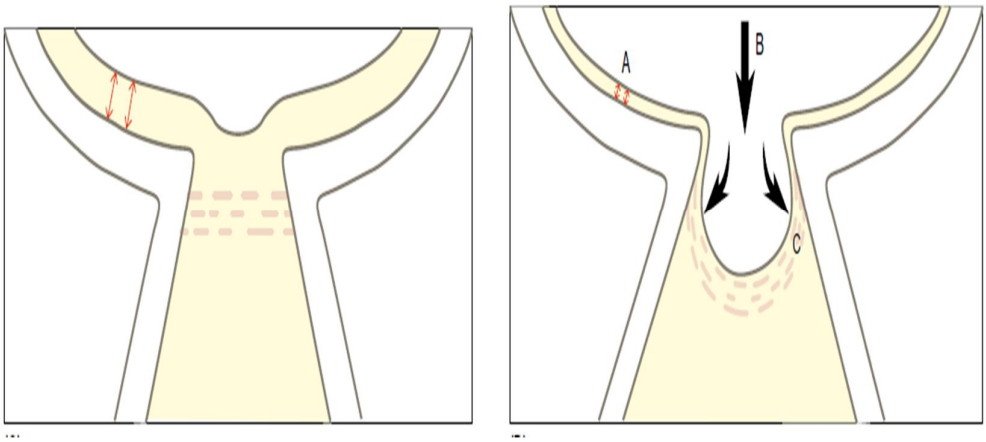
The neuroretinal rim is normally pink in colour.The rim is broadest in the inferior disc, then the superior disc, then the nasal disc, and thinnest in the temporal disc and it correlates with the morphology of the lamina cribrosa – the largest pores and the least amount of interpore connective tissue are in the inferior and superior poles, compared with the nasal and temporal sectors.

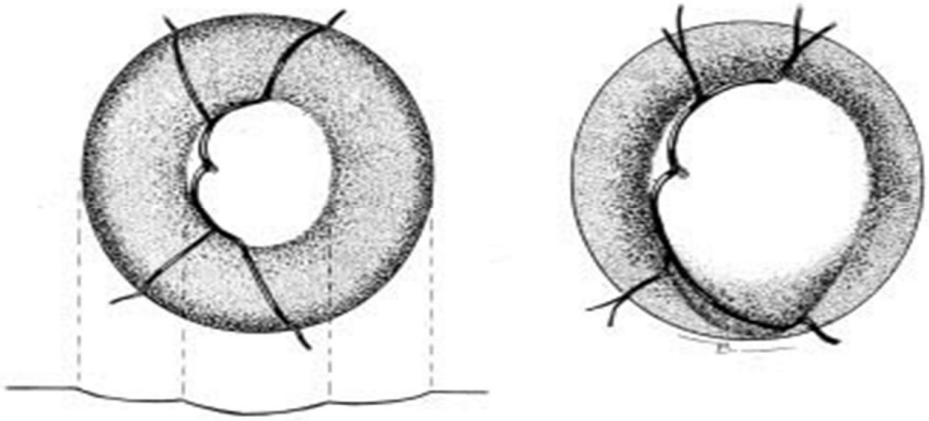
OPTIC NERVE HEAD IN GLAUCOMA

1. Progressive enlargement and/or deepening of the cup (‘cupping’ is the direct result of axonal loss and the resulting structural alterations in the lamina cribrosa ie backward bowing lamina cribrosa)[31]

2. Neuroretinal rim is formed by the extension of the superficial nerve fiber layer into the optic disc and hence it is important to evaluate neuroretinal rim. NRR changes in glaucoma are notching of the rim, NRR loss and thinning in one or more quadrants, asymmetry of NRR between the two eyes and haemorrhages crossing the rim.

OPTIC DISC CUPPING AND NRR THINNING





3. VASCULAR SIGNS

- " Bayonneting," occurs when a vessel take a sharp 90° bend as it passes through an acquired pit formed as a consequence of neuroretinal rim loss and then emerges out from the edge of optic disc.

- "Baring" of circumlinear vessels

- "Nasalization" of blood vessels – Normally blood vessels at the optic nerve head emerge centrally, in glaucoma owing to diffuse neuroretinal rim loss ,major vessels appear to emerge more nasally and this appearance is named as nasalisation of major blood vessels.

- Splinter haemorrhages in the margin of ONH. These hemorrhages are more common in eyes with norm al-tension glaucoma, usually indicating progression of disease.

4. NERVE FIBRE LAYER DEFECTS

Nerve fiber layer defects appear as a series of striations radiating in an arcuate fashion from optic nerve head above and below macula and not crossing the horizontal raphe.

Localized defects – These are wedge shaped areas occurring wider peripherally radiating from the optic nerve head.

Diffuse loss – Diffuse loss of nerve fiber layer is seen in advanced glaucoma.

5. PERIPAPILLARY ATROPHY

Atrophy around the disc consists of two zones –

- Beta zone - Zone of chorioretinal atrophy immediately surrounding the disc marked by significant atrophy of the retinal pigment epithelium and

choriocapillaris, with more clear visibility of the choroidal vessels and sclera.

- Alpha zone - alpha zone surrounds the beta zone and represents areas of chorioretinal thinning occurring as irregular areas of hypo and hyperpigmentation.

OPTIC NERVE HEAD ASSESSMENT

DIRECT OPHTHALMOSCOPY

It is the most commonly used method for assessing the optic nerve head by general ophthalmologists and it provides a non stereo view of the optic nerve head.

SLIT LAMP BIOMICROSCOPY

Optic nerve head can be assessed with a slit lamp biomicroscopy using hand held high power convex lenses (78 D & 90 D) and fundus contact lenses like Goldmann three mirror lens to provide a stereoscopic view of the disc

FUNDUS PHOTOGRAPHY

Fundus photography is the most widely used technology nowadays to document the optic nerve head appearance objectively and also helps in detecting the progression of the disease.

CONFOCAL SCANNING LASER OPHTHALMOSCOPY

Confocal scanning laser ophthalmoscopy takes transaxial laser scans (around 64) through the optic nerve head and peripapillary retina to reconstruct a high resolution three dimensional imaging of optic nerve head and nerve fiber layer.

Heidelberg Retinal Tomograph (HRT) is a prototype of Confocal scanning laser ophthalmoscopy.

Advantage – Rapid image acquisition time, reduced need for pupillary dilation or clear media.

OPTICAL COHERENCE TOMOGRAPHY

OCT uses low-coherence laser light (850 nm) and based on its property of optical backscattering,it takes axial cross-section of tissues as it passes through layers of different optical density. OCT assess optic nerve head using this laser interferometry principle and quantifies the amount of nerve tissue in optic nerve.

NERVE FIBER LAYER ANALYSIS

SCANNING LASER POLARIMETRY (GDxVCC)

In Scanning laser polarimetry ,a laser beam is passed to the posterior retina and changes in the polarization of the reflected beam is measured and is used to quantify peripapillary RNFL .The software used in nerve fiber analyzer is named as GDx. VCC stands for variable corneal compensator which accounts for the variable corneal birefringence.

OPTICAL COHERENCE TOMOGRAPHY

Optical coherence tomography uses the reflected and backscattered light to create reflected images of various retinal layers and is displayed as quadrants, clockhours and overall mean as measured by computer analysis.

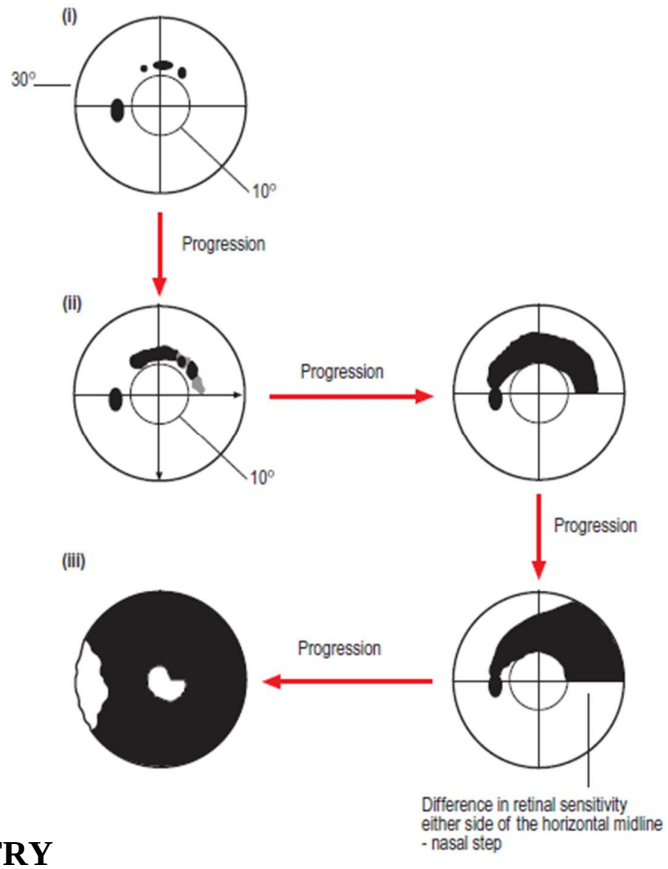
Fourier-domain OCT yields up to five times higher resolution with faster imaging speed (60 times faster) than conventional time-domain OCT.

VISUAL FIELDS

Visual field testing gives a non-invasive direct measure of visual function. The distribution and arrangement of the retinal nerve fibres govern the pattern of visual field loss in glaucoma and scotomas follow the shape of these nerve fibre bundles, giving rise to characteristic glaucomatous field defects which may be absolute, or relative, or a combination of both.

GLAUCOMATOUS VISUAL FIELD DEFECTS

- Early visual loss within the arcuate area (starts from blind spot and arches above or below fixation), they most often appear as localised defect or PARACENTRAL SCOTOMAS
- Early arcuate defect may connect with the blind spot giving rise to SIEDEL SCOTOMA.
- The localised defect then coalesce filling the entire arcuate area from blind spot forming ARCUATE OR BJERRUM SCOTOMA
- Further disease progression leads on to DOUBLE ARCUATE OR RING SCOTOMA.
- Loss of nerve fibers rarely proceeds at the same rate in the upper and lower portions of the horizontal meridian creating a step like defect along the horizontal raphe referred to as the NASAL STEP.
- Continued damage results in a temporal island and central island in advanced glaucoma.[33]
- Temporal island is more resistant and may persists lond after central vision is lost.



PERIMETRY

There are two main types of perimetry namely

1. Kinetic perimetry
2. Static perimetry

KINETIC PERIMETRY

A stimulus of given size and brightness is selected and moved from where it is not seen until its threshold is crossed. Island of vision is kinetically explored along XY axis. Eg – Goldmann kinetic perimetry.

STATIC PERIMETRY

Automated static perimetry is the most reliable and sensitive means of testing a visual field. Here relative differential light sensitivity throughout the field of vision is determined based on either age corrected normal data or patients response to preliminary spot tests.

Testing Strategies:

1.Suprathreshold:

In this strategy a stimulus that is slightly brighter than the anticipated normal for the corresponding retinal location is presented. It is mainly used in screening of glaucoma.

2.Threshold:

A staircase procedure is applied in which a retinal threshold is crossed by increasing or decreasing stimulus and is then crossed second time with smaller increments of change in luminosity.

Types of threshold testing :

1. Standard Full Threshold Testing

The differential light sensitivity is determined at 76 locations spread over a central 30 degrees at 6 degrees apart using staircase

phenomenon.Here the threshold is crossed twice , initially in 4dB increments followed by 2 dB increments.

2. Fastpac

Threshold is estimated with single crossing in 3 dB in contrast to standard double threshold crossing with 4dB and 2 dB, so as to reduce time

3. Swedish Interactive Threshold Algorithm (SITA)

Swedish Interactive Threshold Algorithm (SITA) uses standard 24-2 or 30-2 patterns to assess visual field based on the probability analysis of patterns of glaucomatous damage .It reduces testing time to half and is more time efficient than standard full threshold strategy.

There are two versions off SITA currently available now namely

SITA Standard

SITA Fast

SITA standard and SITA Fast are so fast that they take approximately half the time to complete as compared to standard full threshold and FASTPAC algorithm respectively.

STANDARD ACHROMATIC PERIMETRY (SAP)

Also known as white on white perimetry , here white stimulus is projected on white background. The target is presented randomly at 54 locations within the central 24 degree or 76 locations in central 30 degree.

SHORT WAVE AUTOMATED PERIMETRY(SWAP)

Short wave automated perimetry uses blue wave length as stimulus and a specific colour and brightness of yellow is used as back ground illumination. The red and green cones are desensitised by yellow back ground. The 440nm blue stimulus falls on blue cones.

Blue yellow ganglion cells are selectively damaged in early glaucoma hence early diagnosis is possible by SWAP by 3-5 years earlier than standard achromatic perimetry.

FREQUENCY DOUBLING PERIMETRY

Frequency doubling perimetry is designed based upon a frequency doubling illusion that occurs when viewing a grating with a low spatial frequency and a high temporal rate. The target in frequency doubling perimetry is a sinusoidal grating (0.25 cycles per degree)that subtends an angle of 10 degree. Early glaucomatous defect are detected early than standard perimetry and has a advantage of shortest time,portability and reproducibility.

PRIMARY OPEN ANGLE GLAUCOMA

The mechanistic classification is probably the most common scheme for sorting out the various glaucomatous diseases. The main division of this classification is open-angle and angle closure glaucoma based on gonioscopic findings. The other important differentiation in this classification scheme is between primary (primary disease of the eye with no associated conditions or diseases) and secondary glaucomas (where the glaucoma is attributed to some underlying condition or disorder).

Primary open-angle glaucoma is an optic neuropathy, which is chronic and progressive in nature. It is usually a bilateral and asymmetrical disease of unknown cause where optic nerve is damaged by IOP and other unknown factors leading on to loss of ganglion cells of the retina.

Idiopathic open-angle glaucoma,chronic open-angle glaucoma (COAG) and chronic simple glaucoma are the other synonymous terms used in context with primary open angle glaucoma.

Three elements are needed to make the diagnosis POAG

(1) Anterior chamber angles must be ‘open’ by gonioscopy

(2) There must be no known secondary cause for the glaucoma

(3) There must be optic nerve damage, manifested by optic disc changes or nerve fiber layer changes consistent with glaucoma or characteristic visual field abnormalities.

It is most notable that intraocular pressure is not part of the definition but increased IOP is a risk factor associated with the development of disease and is not the disease itself.

TERMINOLOGY

Normal-Tension Glaucoma

Normal tension glaucoma is one where patients have optic disc and visual field changes suggestive of glaucoma but their IOP is always below 21mmHg. Such patients with the above said findings with a open anterior chamber angle are said to have normal tension glaucoma.

Ocular Hypertension

Patients who have an IOP consistently above 21 mm Hg for which there is no apparent cause with normal optic nerve head and visual fields are said to have ocular hypertension.

Glaucoma suspect

A person with at least one of the following findings in either eye is named as Glaucoma Suspect.

- Elevated IOP over 22 mmHg.
- Glaucomatous Optic nerve head changes or nerve fiber layer defect.
- Visual field abnormality consistent with glaucoma.

EPIDEMIOLOGY

- In 2010, it is estimated that glaucoma affects approximately 60.5 million people worldwide.
- There were 44.7 million people with primary open-angle glaucoma of the total predicted 60.5 million people with glaucoma.
- Glaucoma in India:

In India 11.2 million people were affected with glaucoma, among them POAG is estimated to affect 6.48 million persons in 2010.

PATHOPHYSIOLOGY

Two pathophysiological issues to be addressed in primary open angle glaucoma includes:

(1) Pathophysiology of elevation of intraocular pressure.

(2) Pathophysiology of cupping and atrophy of the optic nerve.

PATHOPHYSIOLOGY OF INCREASED INTRAOCULAR PRESSURE:

Elevated IOP in open angle glaucoma occurs secondary to structural alterations in the aqueous outflow channels. Juxtacanalicular tissue with its

greatest phagocytic activity and high mucopolysaccharide content is considered as the main site of aqueous outflow resistance.

Mechanics of outflow obstruction have been explained by various theories . Some of them are as follows:

1. Materials like glycosaminoglycans, amorphous material, red blood cells, extracellular lysosomes ,pigment, protein etc can get deposited and cause obstruction of the trabecular meshwork.

2. A normal constituent which is synthesized excessively or not catabolized properly can also obstruct the meshwork. Added on to it normal phagocytic activity is lost in this type of glaucoma trabecular meshwork to self clean itself leading on to increased resistance.

3. Giant vacuoles found in the the inner wall of Schlemm's canal has an important role in maintenance of IOP and their reduction in size and number seen in open angle glaucoma is thought to be responsible for elevated IOP.

4. Trabecular endothelial cells are involved in functions like synthesis and degradation of macromolecules and phagocytosis. These functions are lost when there is loss of trabecular endothelial cells and lead on to glaucoma.

HISTOPATHOLOGY

Histopathologic studies of the aqueous outflow pathway in patients with primary type of open angle glaucoma has revealed abnormalities like fused trabecular beams,fragmentation and long-spacing of collagen in the trabecular beams, decreased number of trabecular endothelial cells, decreased number of giant vacuoles in the inner wall of schlemn's canal, narrowing or collapse of schlemm's canal, thickened scleral spur etc.

Despite extensive research the precise mechanism of outflow obstruction in this condition cause of the still remains unclear.

PATHOPHYSIOLOGY OF GLAUCOMATOUS OPTIC NEUROPATHY

Features characteristic of glaucomatous optic neuropathy includes:

- Characteristic cupping of the optic disc
- Apoptosis of retinal ganglion cells and their axons.

The mechanisms of glaucomatous damage and the initial site of involvement is not well understood till date. It is thought that multiple factors and their complex interplay lead on to glaucoma rather than each factor acting

individually .Final end result will be permanent visual loss as a result of retinal ganglion cell death.

Various theories have been put forth to explain the pathophysiology of ganglion cell death namely

- Backward bowing of the lamina cribrosa cause mechanical kinking of the axons as they exit through the lamina pores.This in turn lead to focal ischemia, interfere with axoplasmic flow ,deprive the axons of neurotrophins and trigger death of axons.
- Vascular theories propose that cell death is triggered by ischemia.
- Genetic theories propose that cell death is triggered by genetic predisposition.

Following the death of individual axons, substances may be released into the environment that causes a secondary triggering of apoptosis in neighboring cells, including glutamate (a neurotransmitter that may cause excitotoxicity), calcium, nitric oxide, and free radicals.

RISK FACTORS

Risk factors involved in the occurrence of primary open angle glaucoma can be grouped as

Demographic factors

Ocular factors

Systemic factors

DEMOGRAPHIC RISK FACTORS

AGE

POAG is becomes more prevalent as age advances and is seen most often in patients around 60 years of age (and rarely before 40 years old). Prevalence tends to roughly double for each decade over 40 years and is about 10 fold higher above 80 years of age.[8,9,10]

GENDER

There is discordance among various studies about gender association in glaucoma. Framingham study[62] , studies by Ramakrishnan et al [9], Leske et al etc have reported increased prevalence of POAG in males whereas Blue mountain eye study , St lucia ,Andhra Pradesh eye study etc have reported increased prevalence in females. Some studies found no significant association.

RACE

POAG is more common in people of African ancestry and the disease tends to more severe in them.

FAMILY HISTORY

Family history of glaucoma is associated with an increased risk of POAG. Genetic influence occurs through polygenic or multifactorial transmission. Recent studies have demonstrated the association of juvenile-onset open-angle glaucoma with GLC1A gene located on chromosome 1 in the q23–25 region and adult-onset open-angle glaucoma with GLC1B gene located on chromosome 2.

OCULAR RISK FACTORS

INTRAOCULAR PRESSURE

Intraocular pressure is considered to be the major and treatable risk factor involved in the development and worsening of glaucoma. Prevalence of nerve damage is higher among individuals who have pressures of 25-30 mm Hg.

CENTRAL CORNEAL THICKNESS

Central corneal thickness has been shown in many recent studies, not only to affect the accuracy of intraocular pressure measurements, but also to perhaps act as its own intrinsic risk factor for glaucoma damage.There is increased risk of conversion from ocular hypertension to open-angle glaucoma in patients with thin corneas. People of African ancestry had thinner corneas and this is explained as the factor responsible for increased risk for conversion from ocular hypertension to open-angle glaucoma among black population.

MYOPIA

Many studies have associated myopia with open angle glaucoma but the mechanisms are not fully explained, increased IOP and large cup disc ratioseen in myopic patients is thought of as a risk factor.[46]

SYSTEMIC RISK FACTORS

DIABETES MELLITUS

The prevalence of POAG have been reported to be higher in diabetic population in many population based studies.[14,64,65]

HYPERTENSION

Hypertension may contribute to the risk of development of glaucoma through vascular mechanisms and increased IOP occurring with increased systemic blood pressure may also play a role.

VASOSPASM

Associations between vasospastic phenomenon (cold extremities, migraine, Raynaud's syndrome) and glaucoma have led to an ischaemic hypothesis for glaucoma.

SMOKING

Blue mountain eye study reported a small increase in IOP in smokers but other studies have reported no difference in the prevalence of POAG among smokers and non smokers.

HYPERTENSION AND PRIMARY OPEN ANGLE GLAUCOMA

There are a number of risk factors involved in the development and progression of primary open angle glaucoma but treatment for glaucoma till date is targeted towards IOP reduction. But despite sufficient IOP reduction, disease continue to progress and many patients suffer ongoing vision loss, this

explains the fact that there are risk factors other than IOP which can also cause disease development and progression. Risk factors like advancing age, positive family history and ethnicity (especially African ancestry) have long been associated with glaucoma. Recently risk factors which alter the blood supply of the optic nerve head like systemic HT, atherosclerosis and vasospasm have been evaluated. Evaluation of the role of systemic hypertension in the development and progression of primary open angle glaucoma has attracted attention in recent years as it represents a potentially modifiable risk factor and thus provides option for new treatment strategies beyond IOP reduction.

OPTIC NERVE HEAD PERFUSION

Blood flow to the optic nerve head is given by the formula

Blood flow = Ocular perfusion pressure / vascular resistance

OCULAR PERFUSION PRESSURE

Blood flow to the capillary network of the lamina cribrosa region of the optic nerve can be described by a parameter known as ocular perfusion pressure (OPP). Ocular perfusion pressure is given by the formula i.e

$$OPP = BP - IOP.$$

This relationship between IOP and blood pressure is important clinically because glaucoma and hypertension often co-exist in ageing populations.

Abnormalities in blood flow and the ability of the eye to maintain its blood flow against changes in perfusion pressure are central to the pathogenesis of glaucoma. When the capacity of the eye to maintain blood flow in the face of variations of OPP by a process called autoregulation decreases, the risk of impaired oxygen and nutrients supply to the eye increases and this in turn can result in neuronal dysfunction.

BLOOD PRESSURE AND INTRAOCULAR PRESSURE

Many population based studies have proved an association between high blood pressure and IOP . Each 10 mmHg rise in systolic blood pressure is associated with but only a small increase in IOP (approximately 0.27 mmHg). The physiological basis of this relationship between blood pressure and IOP remains unclear.

Various hypotheses has been proposed for the same

- Both elevated IOP and blood pressure might be driven by a common extrinsic factor such as an age-related increase in the sympathetic tone.

- elevated BP rises ciliary artery pressure which in turn results in an increase of ultrafiltration component of aqueous production, resulting in IOP elevation.

- Small increase in venous pressure occurs as arterial pressure increases, this increase in venous pressure reduces aqueous clearance resulting in higher IOP.

BLOOD PRESSURE AND VASCULAR RESISTANCE

Vascular resistance in a circulation is directly related to the state & calibre of the vessels in the circulation which in turn depends on many factors like autoregulation, vessel wall changes like arteriosclerosis etc.

Blood pressure can influence vascular resistance by various mechanisms.

- There is increased vascular resistance in terminal arterioles all over the body, the basic pathology in hypertension.

- Autoregulation is the inherent ability of an organ to maintain constant blood flow despite changes in perfusion pressure. Systemic hypertension causes endothelial cell damage/ dysfunction and abnormal release of vasoactive substances resulting in an alteration of autoregulatory mechanism.

- The vascular endothelial cells release various known endothelial vasoactive agents of which nitric oxide (NO; vasodilation) and endothelin-1 (ET-1; vasoconstriction) are perhaps the most important factors and have opposing actions in ocular blood flow.

- Abnormalities in endothelial derived vasoactive agents particularly reduction in production of NO can occur in hypertension

- Higher systemic concentrations of catecholamines and circulating vasoconstrictor agents like angiotensin in arterial hypertension can have a direct effect on ONH blood flow.

So changes in blood flow and blood pressure play a vital role in the development and progression of glaucoma but the mechanisms involved are not fully understood till date.

OBJECTIVES OF THE STUDY

- To determine the prevalence of primary open angle glaucoma in patients with systemic hypertension.
- To find the association of primary open angle glaucoma, intraocular pressure and systemic hypertension.

MATERIALS AND METHODS

This cross sectional case control study was conducted at Stanley medical college from November 2012 to October 2013.Patients with documented history of hypertension and on antihypertensive medication attending hypertensive clinic at Stanley medical college were included in the study. Informed consent was obtained from all patients.

Sample size – 100 hypertensive patients and 100 age-sex matched non hypertensive controls.

INCLUSION CRITERIA

1. Patients with documented systemic hypertension on antihypertensive medications.

2. Patients above 40 yrs of age

3. Age and sex matched non hypertensive controls in the ratio of 1:1

EXCLUSION CRITERIA

1. Patients less than 40 yrs of age

2. Patients with secondary glaucoma

3. Patients with diabetes and patients with family history of glaucoma.

4. Myopia

5. Corneal scarring/ opacity on which gonioscopy is not helpful.

6. Previous ocular trauma or diseases other than primary open angle glaucoma.

METHODS

Patients with systemic hypertension and age and sex matched controls meeting the criteria mentioned above were included in the study after taking informed consent. A detailed history regarding past medical illness including hypertension, duration of hypertension, diabetes mellitus, family history of glaucoma, myopia was taken. Details regarding antihypertensive medications and any other medications that the patient was on were also obtained.

Patients with family history of glaucoma, diabetic patients with a history of diabetes or on treatment for the same or with an elevated fasting blood

glucose level of >140 mgs % [61] and myopic patients with a spherical equivalent refractive error > 1.00 diopter were excluded from the study.

Systemic Hypertension was defined by documented diagnosed case of hypertension with a blood pressure of ≥140/90 and on treatment with anti hypertensive medications.

Primary open angle glaucoma was defined by the presence of open angles in gonioscopy with any two of the following features;

IOP >21 mm Hg

Glaucomatous optic disc changes

Visual field changes suggestive of glaucoma.

All participants underwent detailed examination. Assessment included:

- Systemic Blood Pressure recording using mercury sphygmomanometer with average of 3 readings at an interval of 10 min in right upper limb in supine position
- Visual Acuity was assessed using Snellens chart.
- Slit lamp examination to evaluate anterior segment.
- Refraction

- Gonioscopy was done with Goldmann's single mirror goniolens and angle was graded according to Modified Shaffer's grading.

- IOP measured using Goldmann's applanation tonometer.

- Visual fields assessed using octopus perimetry.

- Fundus examination by direct, indirect ophthalmoscopy followed by +90D assessment of optic nerve head with slit lamp.

- Laboratory investigations – Fasting blood sugar levels.

OBSERVATION AND RESULTS

A total of 100 patients with systemic hypertension and 100 age & sex matched controls were studied. Patients were divided accordingly into two groups,

Group 1 – with systemic hypertension, Group 2 – without systemic hypertension, for the purpose of determining the prevalence of primary open angle glaucoma in the two groups and analysing the statistical significance.

The statistical analysis was performed with SPSS (statistical analysis of social science) Version 16. Tests for statistical significance were done using Annova test. *P* value of less than 0.05 was considered significant. The numbers of glaucoma patients with hypertension were compared to the number of control subjects with the same conditions using the Fisher's exact test and odds ratio calculated.

AGE DISTRIBUTION

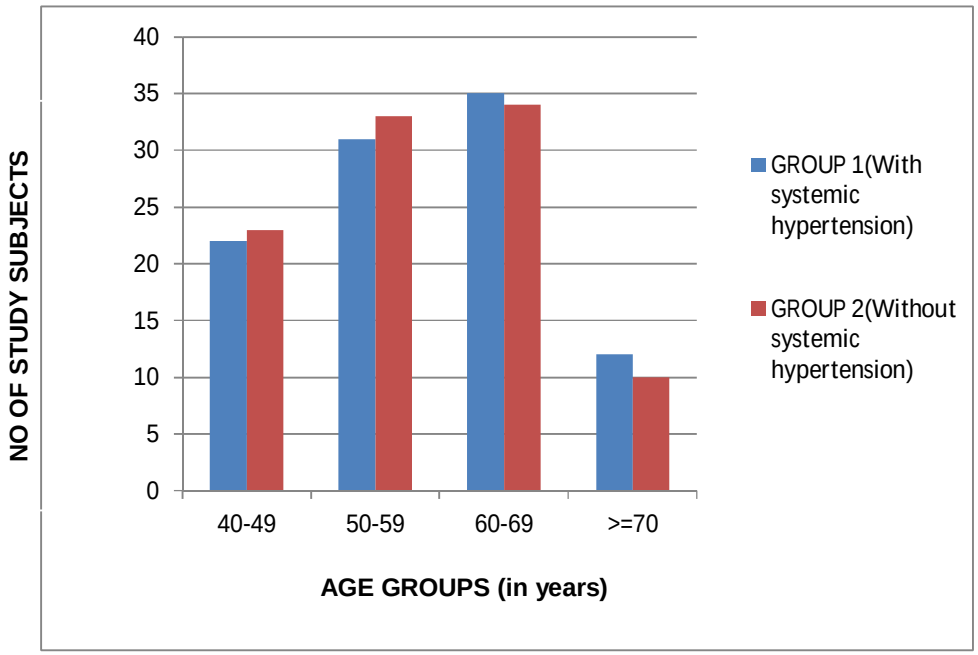
The study population (200) consists of patients in the age group of 40 to 80 years. In group 1, there were 22 patients in the age group of 40-49 years, 31 patients in the age group of 50-59 years, 35 patients in the age group of 60-69 years and 12 patients were above 70 years of age. In group 2, there were 23 patients in the age group of 40-49 years, 33 patients in the age group of 50-59 years, 34 patients in the age group of 60-69 years and 10 patients were above 70 years of age.

Age distribution within groups is given in table 1. The mean age of GROUP 1 is 57.94 years and mean age of GROUP 2 is 57.55 years.

TABLE 1: AGE DISTRIBUTION OF STUDY SUBJECTS

AGE GROUPS (in years)	**GROUP 1 (with systemic hypertension)**	**GROUP 2 (without systemic hypertension)**	**TOTAL**
40 -49	22	23	45
50 – 59	31	33	64
60 -69	35	34	69
≥70	12	10	22
TOTAL	**100**	**100**	**200**

FIGURE 1: AGE DISTRIBUTION OF STUDY SUBJECTS



AGE DISTRIBUTION OF PATIENTS WITH GLAUCOMA

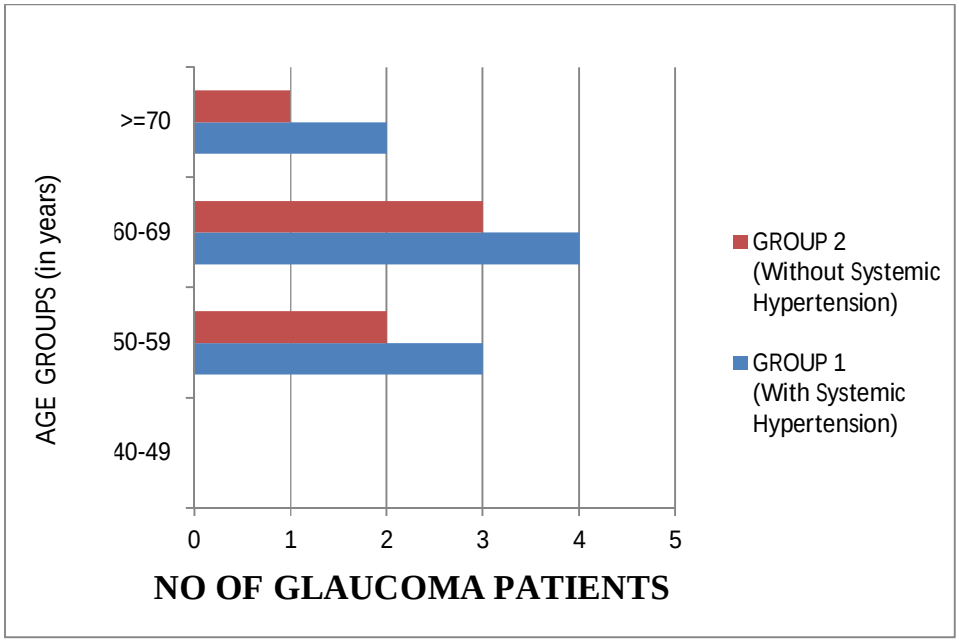
There were 9 patients (9/100) with glaucoma in group 1 and 6 patients (6/100) with glaucoma in group 2.Among these 15 patients with glaucoma, 5 were in the age group of 50-59 years, 7 were in the age group of 60-69 years and 3 were above 70 years of age.

Among 15 patients with POAG – 33.3 % (5/15) were below 60 years of age and 66.6% (10/15) were above 60 years of age. The mean age of patients with glaucoma was 62.6 years.

TABLE 2: AGE DISTRIBUTION OF GLAUCOMA PATIENTS

AGE GROUPS (in years)	GLAUCOMA PATIENTS		TOTAL
	GROUP 1 (with systemic hypertension)	GROUP 2 (without systemic hypertension)	
40 -49	-	-	-
50 – 59	3	2	5
60 -69	4	3	7
≥70	2	1	3
TOTAL	**9**	**6**	**15**

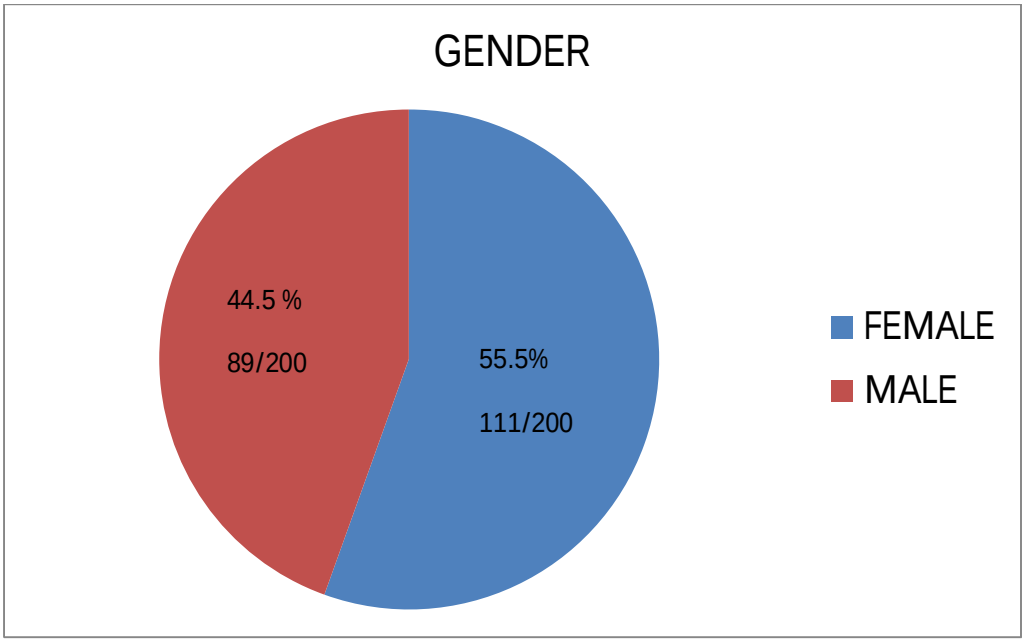
FIGURE 2: AGE DISTRIBUTION OF GLAUCOMA PATIENTS



GENDER DISTRIBUTION

There were 44 males and 56 females in group 1 and in group 2 there were 45 males and 55 females. The study subjects (200) in total consisted of 89 males (89/200) and 111 females (111/200) i.e. 44.5 % were males and 55.5 % were females.

FIGURE 3: GENDER DISTRIBUTION OF STUDY SUBJECTS



PREVALENCE OF POAG IN DIFFERENT GENDERS

POAG was present in 5 males and 4 females in group 1 and in 4 males and 2 females in group 2.Among 15 (15/200) patients with POAG, 9 were males (9/15) and 6 were females (6/15). There was no statistically significant difference in the gender distribution of POAG patients.

P Value – 0.60.

TABLE 3: GENDER DISTRIBUTION OF POAG PATIENTS

GENDER	GROUP 1 (with systemic hypertension	GROUP 2 (without systemic hypertension)	TOTAL
MALE	5	4	9
FEMALE	4	2	6
TOTAL	**9**	**6**	**15**

BLOOD PRESSURE

The mean systolic and diastolic blood pressure of the study subjects were as follows:

POAG PRESENT:

HYPERTENSIVE GROUP:

SYSTOLIC = 143.11 mmHg

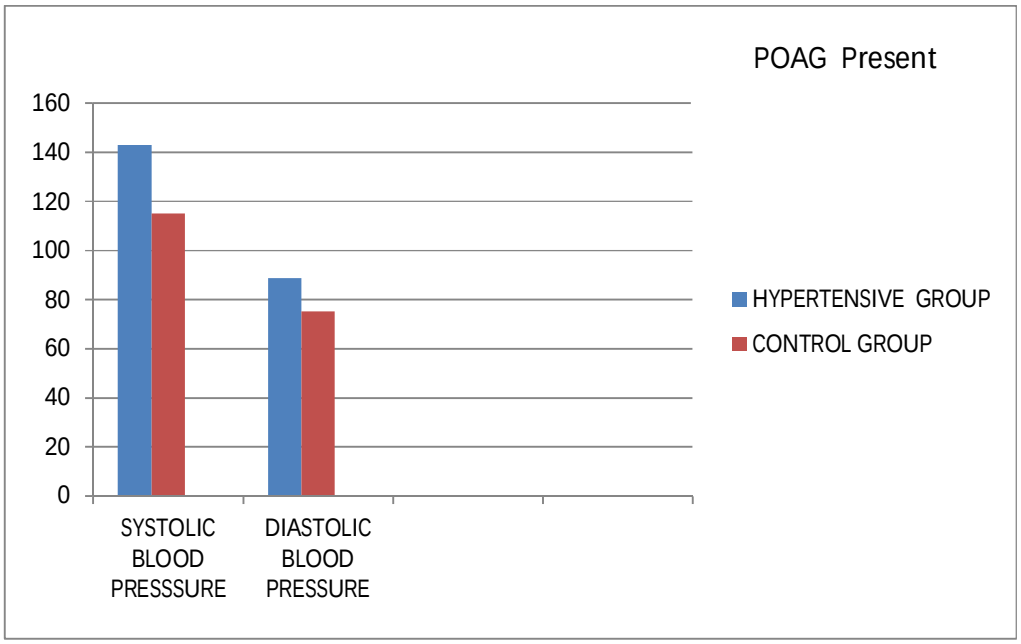
DIASTOLIC = 88.89 mmHg

CONTROL GROUP:

SYSTOLIC = 115 mmHg

DIASTOLIC = 75 mmHg

FIGURE 4: BLOOD PRESSURE IN PATIENTS WITH POAG



POAG ABSENT:

HYPERTENSIVE GROUP:

SYSTOLIC = 138.52 mmHg

DIASTOLIC = 88.13 mmHg

CONTROL GROUP:

SYSTOLIC = 116.59 mmHg

DIASTOLIC = 74.57 mmHg

FIGURE 5: BLOOD PRESSURE IN PATIENTS

WITHOUT POAG

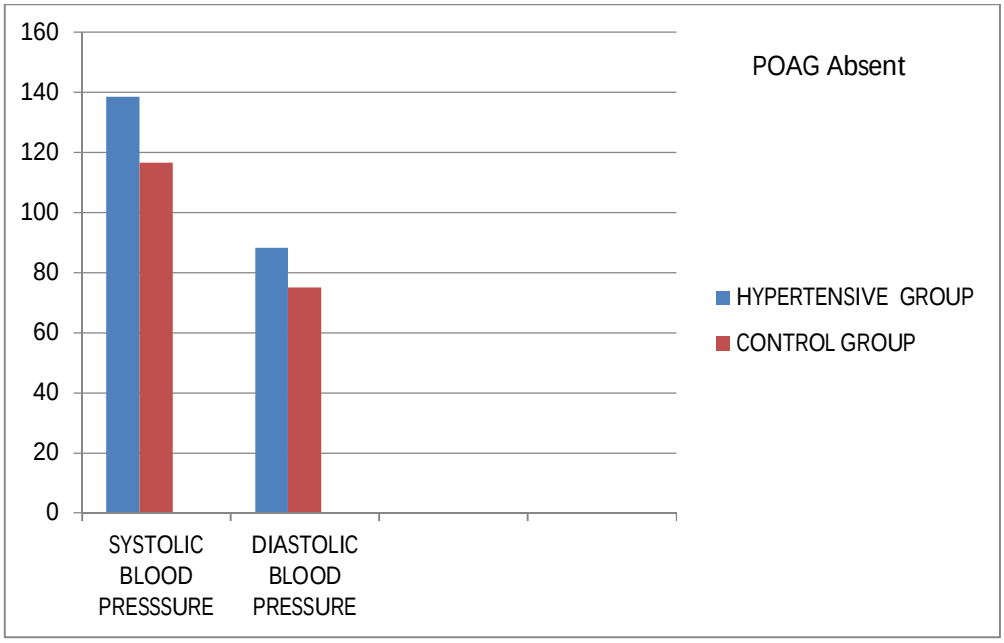


TABLE 4:BLOOD PRESSURE AND POAG

GROUP	POAG PRESENT		POAG ABSENT	
	SYSTOLIC BLOOD PRESSURE	DIASTOLIC BLOOD PRESSURE	SYSTOLIC BLOOD PRESSURE	DIASTOLIC BLOOD PRESSURE
HYPERTENSIVE GROUP	143.11	88.89	138.52	88.13
CONTROL GROUP	115	75	116.59	74.57

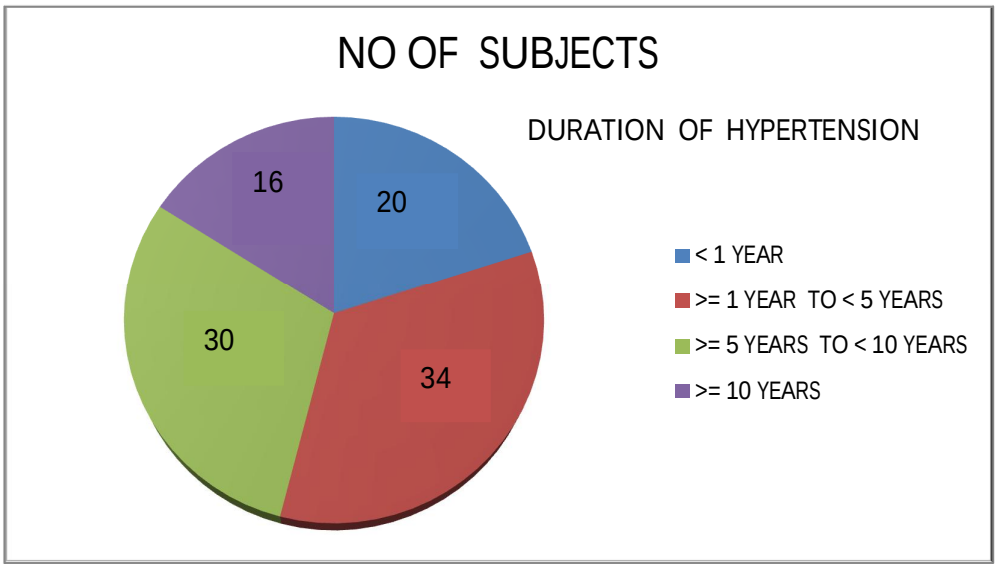
DURATION OF HYPERTENSION

Duration of hypertension of the 100 hypertensive subjects was grouped as follows:

TABLE 5: DURATION OF HYPERTENSION

DURATION OF HYPERTENSION	NO OF SUBJECTS
< 1 YEAR	20
≥1 YEAR TO < 5 YEARS	34
≥ 5 YEARS TO < 10 YEARS	30
≥ 10 YEARS	16
TOTAL	**100**

FIGURE 6: DURATION OF HYPERTENSION



DURATION OF HYPERTENSION AND POAG

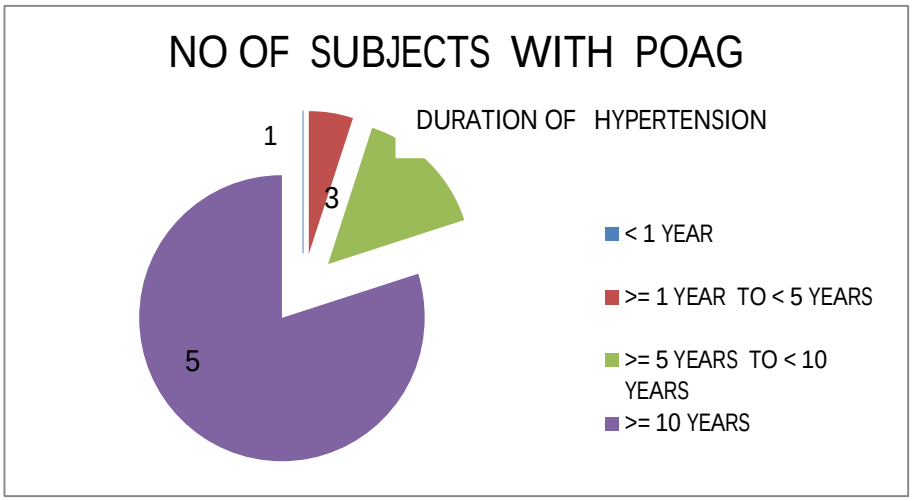
Among glaucoma subjects in group 1, 5 were hypertensive for ≥10 years and the remaining 4 were hypertensive for less than 10 years. Duration of hypertension of glaucoma subjects were as follows:

TABLE 6: DURATION OF HYPERTENSION AND POAG

DURATION OF HYPERTENSION	NO OF SUBJECTS WITH POAG
< 1 YEAR	-
≥1 YEAR TO < 5 YEARS	1
≥ 5 YEARS TO < 10 YEARS	3
≥ 10 YEARS	5
TOTAL	**9**

Out of 9 patients with glaucoma in hypertensive group, 55.5% (5/9) was hypertensive for ≥10 years.

FIGURE 7: DURATION OF HYPERTENSION AND POAG



INTRAOCULAR PRESSURE

Mean IOP in hypertensive group was 17.30 with a standard deviation of 3.268. Mean IOP in control group was 16.02 with a standard deviation of 3.250.

TABLE 7: MEAN IOP OF STUDY SUBJECTS

GROUP	MEAN IOP	
HYPERTENSIVE GROUP	17.30 ± 3.268	**P VALUE =0.006**
CONTROL GROUP	16.02± 3.250	

STATISTICAL ANALYSIS

By applying annova table, there is a statistical significant difference in IOP between the two groups with a P value of 0.006.

GLAUCOMA AND INTRAOCULAR PRESSURE

There were 15 definitive cases of glaucoma meeting the defined criteria for diagnosis of POAG. Nine of them had an IOP above 21 mm Hg and remaining six had optic disc and field changes suggestive of glaucoma but the IOP was < 21 mm Hg.

Among 200 subjects studied, 18 people had IOP above 21 mmHg. 10 of them had POAG & remaining 8 were ocular hypertensive without optic disc or field changes.

TABLE 8 – IOP AND GLAUCOMA

IOP	POAG PRESENT		POAG ABSENT		TOTAL (%)
	GROUP 1 (WITH HT)	GROUP 2 (WITHOUT HT)	GROUP 1 (WITH HT)	GROUP 2 (WITHOUT HT)	
> 21	5	5	5	3	18(9%)
< 21	4	1	86	91	182(91%)
TOTAL	**9**	**6**	**91**	**94**	**200**

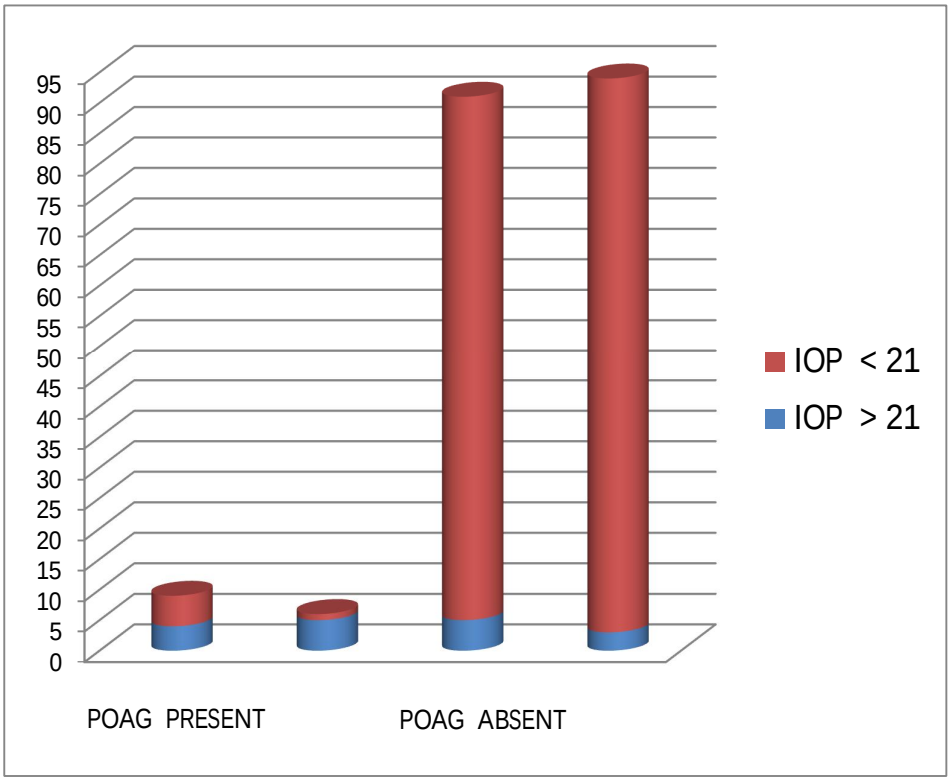
No of glaucoma patients

With IOP > 21mmHg - 10

With IOP < 21 mmHg - 5

Ocular hypertensives - 8

FIGURE 8: INTRAOCULAR PRESSURE AND POAG



SYSTEMIC HYPERTENSION AND POAG

Out of 200 subjects studied, 15 of them was found to have glaucoma amounting to an OVERALL PREVALENCE rate of 7.5%.

% PREVALENCE IN

HYPERTENSIVE GROUP - 9%

NON HYPERTENSIVE GROUP – 6%

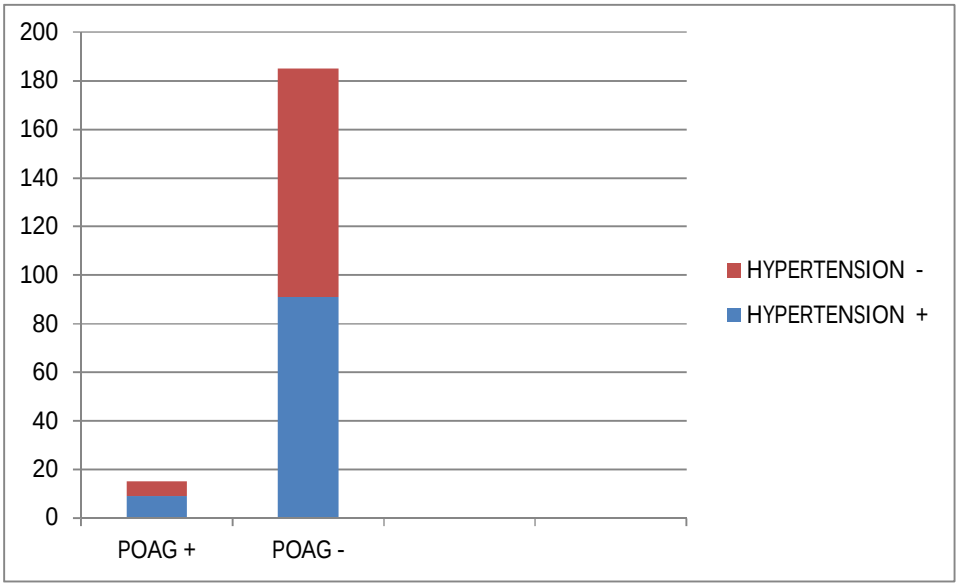
TABLE 9: PREVALENCE OF POAG

RISK FACTOR	POAG Present	POAG Absent	TOTAL
HYPERTENSION +	9	91	100
HYPERTENSION -	6	94	100
TOTAL	**15**	**185**	**200**

STATISTICAL ANALYSIS

Odds ratio and risk estimate was found to be 1.936 with a 95% confidence interval ranging from 0.687 to 5.457 .Odds of hypertensive patient developing POAG was 1.936 which was not statistically significant.

FIGURE 9:HYPERTENSION AND POAG



DISCUSSION

Glaucoma is the most common cause of irreversible blindness in the world and according to WHO estimate, 12.3% of blindness in the world is due to glaucoma. POAG is often asymptomatic characterised by progressive retinal ganglion cell death and visual field loss. IOP still remains the focus of therapy but many patients experience continued progression of disease despite adequate control of IOP. Extensive research is underway to identify other risk factors like vascular factors, genetics and other systemic conditions that may be responsible for such progression. Majority of those with glaucoma remain undetected, hence identification of risk factors will help in screening this high risk population so that the disease can be diagnosed early which in turn can lead to a reduction in visual morbidity associated with glaucoma.

POAG is a multifactorial disease influenced by various factors like age, race, familial predisposition etc. Prevalence of POAG varies in different population with Africans being affected more with higher prevalence rate (4-8%), and the prevalence rate in white population is in the range of 1-3%. The estimated prevalence of POAG in India is between 0.41 % (Vellore Eye Survey)[60] and 2.56 % (Aravind Comprehensive Eye Survey)[19,8] . A total of 200 patients were examined in our study, among them 15/200 had POAG, with a prevalence rate of 7.5 %. This study being a hospital based study with some

amount of referral bias may be a contributing factor for the higher prevalence as compared to other studies which are population based studies.

The mean age of our study population is 57.94 years in hypertensive group and 57.55 years in nonhypertensive group. Among 15 patients with POAG – 33.3 % (5/15) were below 60 years of age and 66.6% (10/15) were above 60 years of age, this suggest that occurrence of POAG increases with increasing age.The mean age of patients with glaucoma in our study was found to be 62.6 years. Similar results were reported in other studies like Latino eye study[63] (Mean age -65.4 yrs), West Bengal eye survey[57] (Mean age 61.7 yrs) Chennai glaucoma study (mean age - 59.9 yrs) and Aravind comprehensive eye survey[9] (Mean age 60.8 yrs).

Our study comprised of 89 males and 111 females, and POAG was diagnosed in 9 males (9/15) and 6 females (6/15) .Many studies done across the world have reported no gender difference in the occurrence of POAG including studies by Vijaya et al [19]and Venkatraman et al from India. But some studies like those by Ramakrishnan et al[9] , Leske et al[21] etc have reported increased prevalence of POAG in males whereas Blue mountain eye study[11] , Andhra Pradesh eye study[10] etc have reported increased prevalence in females. In our study we found no statistically significant gender difference in prevalence of POAG. [P Value - 0.358]

In our study among the patients with glaucoma in hypertensive group 55.5% were hypertensive for atleast 10 years of duration, this shows that duration of hypertension may have an influence on the development of glaucoma probably by the altered autoregulatory and arteriosclerotic changes occurring in long term hypertensives.

There was statistically significant difference [P Value -0.006] in the mean IOP of hypertensive subjects when compared to non hypertensive subjects in our study with the IOP of hypertensive patients being higher. Elevation of IOP with increased blood pressure is an established factor confirmed by various studies. [21,59.]

The relationship between systemic hypertension and POAG is complex and the information available on the association of systemic hypertension with POAG often appears different among different population. Several large epidemiologic studies have been done worldwide to investigate this relationship, the results of which are conflicting. These conflicting results are not surprising because of the racial difference that exists in the occurrence and complications of hypertension and the complex pathophysiological mechanisms involved in the development of primary open angle glaucoma. According to ischemic hypothesis, systemic hypertension in the presence of elevated IOP would be protective as it would increase the perfusion pressure but chronic hypertensives will also have microvascular damage, increased vascular

resistance and defective autoregulation which in turn can reduce ocular perfusion pressure increasing the risk of ischemic damage.

The Egna-Neumarkt[15], Rotterdam[58], and Blue Mountain Eye studies[47] have reported significant associations between systemic BP and POAG in their cross-sectional data, whereas the Barbados Eye Study[21], Early Manifest Glaucoma Trial, Latino eye study[63] etc do not support any association between systemic BP and occurrence of POAG.

In our study we found no statistically significant correlation between occurrence of POAG and systemic hypertension. The odds of hypertensive patients developing POAG was 1.936 with a 95% confidence interval of 0.687 to 5.457 which was not statistically significant. Our results were comparable to Indian studies like Aravind comprehensive eye survey[9] (Odds Ratio -1.0) and Chennai glaucoma study[8] (Odds ratio - 1.02) which also found no association between hypertension and POAG. Studies by Tielsch et al [47][Black subjects: Odds Ratio: 1.24, White subjects: Odds Ratio: 0.73] and Uhm and Shin et al[48] (Odds Ratio=1.19) also found no significant relationship between HT and POAG.

STUDY	**MEASURE OF ASSOCIATION BETWEEN HT & POAG (OR- ODDS RATIO)**	**STATISTICAL SIGNIFICANCE**
Baltimore study - Tielsch	Black subjects: OR: 1.24	Weak correlation

et al	White subjects: OR: 0.73	
Uhm and Shin et al	OR -1.19	Not statistically significant
Aravind comprehensive eye survey	OR -1.0	Not statistically significant
Chennai glaucoma study	OR - 1.02	Not statistically significant
Our study	OR -1.936	Not statistically significant

The present study has some limitations. Our study is a hospital based study with a small sample size as compared to other related studies which are population based studies with a large sample size. Factors such as systemic antihypertensive drugs that the patient was on was not included in the present analysis but are reported to be associated with the development of POAG.

CONCLUSION

Hypertension has been found to be a risk factor for open-angle glaucoma in various studies and there are studies which contradict the same. The results of our study did not show any significant association of systemic hypertension with POAG. Despite the positive correlation between blood pressure and IOP, the actual change in IOP with increasing blood pressure is small. Since it has been established that IOP is a significant risk factor for POAG, it should be measured and accounted for. It is also found that frequency of primary open angle increases with increasing age and duration of hypertension.

It can be concluded that the results are insignificant to consider systemic hypertension as an independent risk factor for glaucoma but persons with long duration of hypertension and advancing age, need to be monitored for high IOP and optic nerve head changes to aid in early diagnosis and also to minimize visual morbidity and blindness due to glaucoma.

A population based study with a large sample size, taking into consideration the antihypertensive medication and nocturnal dips in blood pressure caused by such medication with nocturnal blood pressure monitoring is essential to arrive at a definitive and clear demonstration of the relationship between systemic hypertension and glaucoma.

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DEPARTMENT OF OPHTHALMOLOGY

STANLEY MEDICAL COLLEGE

HYPERTENSION AND PRIMARY OPEN ANGLE GLAUCOMA

PROFORMA

Name : **Address :**

Age :

Sex :

OP No. : **Phone Number :**

History :

Hypertension History :

Hypertension : **Yes** **No**

Duration :

On Treatment : **Yes** **No**

Medications

Glaucoma :

Known Glaucoma patient : Yes No

On Treatment : Yes No

Medications :

Other Co Morbidities :

Past History :

Personal History :

Family History :

Examination :

Pulse Rate :

Blood Pressure : Systolic –

Diastolic -

Ocular Examination : Right Eye Left Eye

Vision :

Distance :

Near Vision :

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Slit Lamp Examination :

Fundus :

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Gonioscopy :

Automated Perimetry :

Other Investigations :

Diagnosis :

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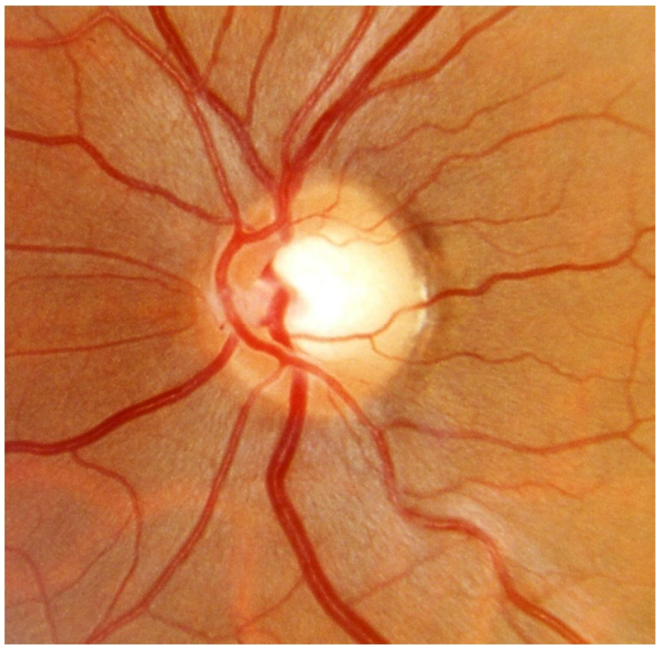

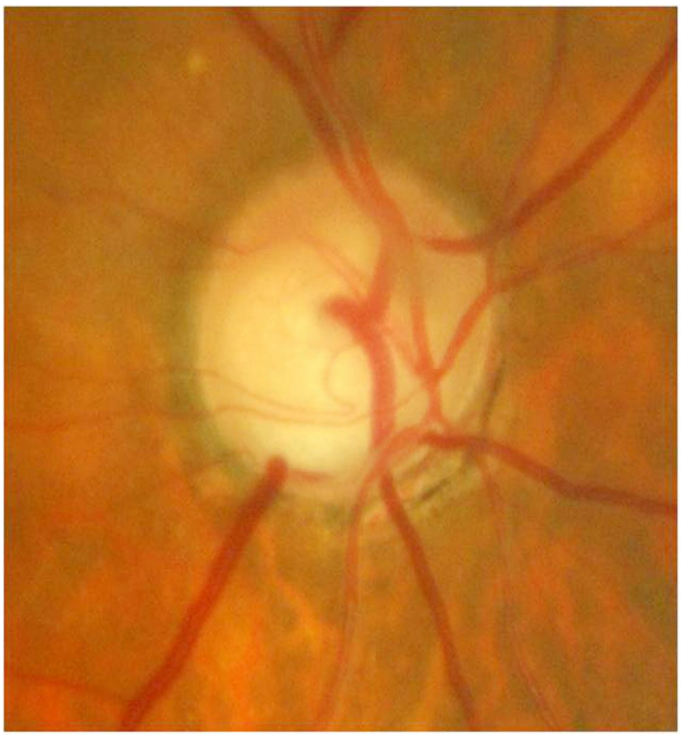

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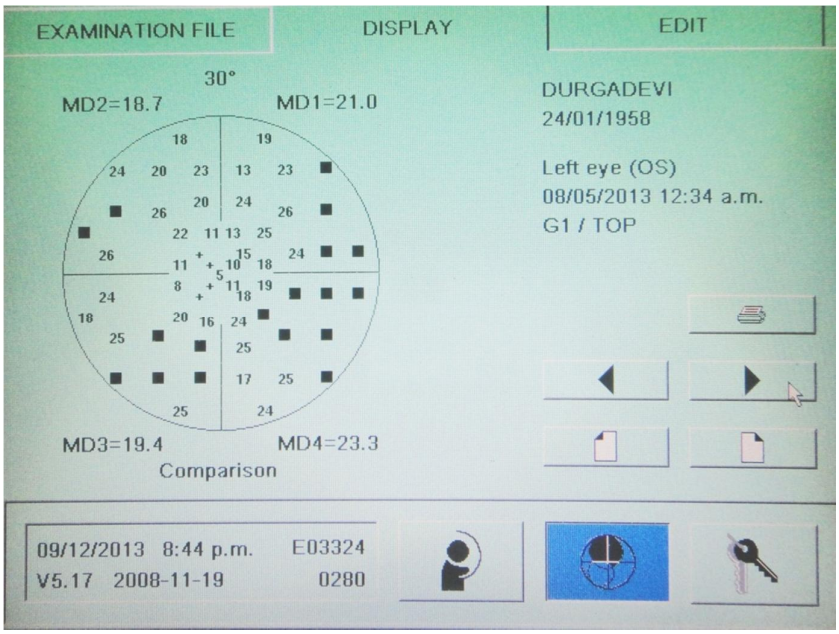
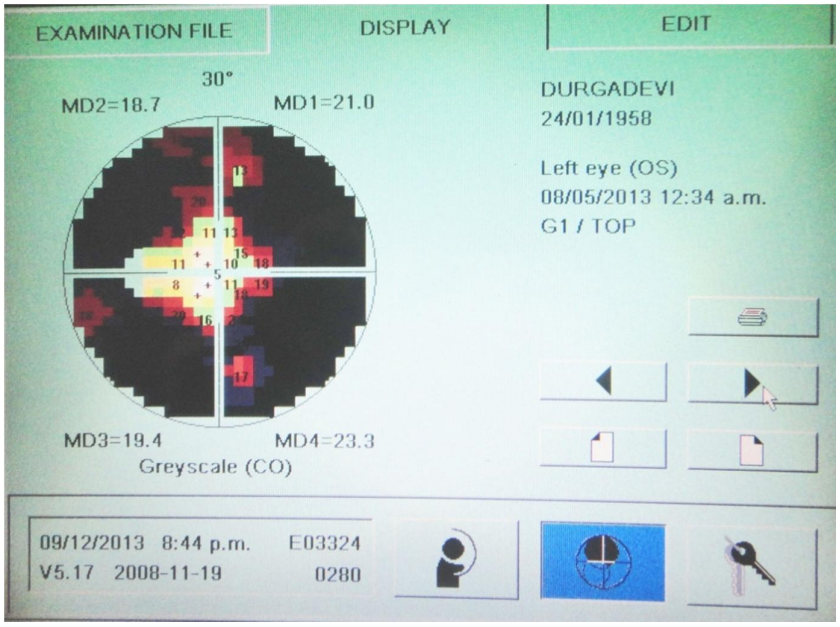
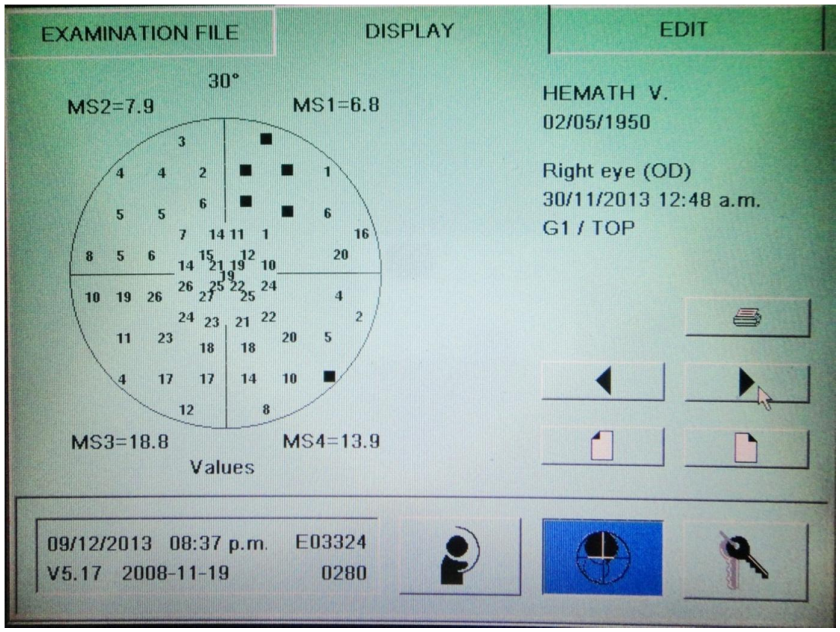
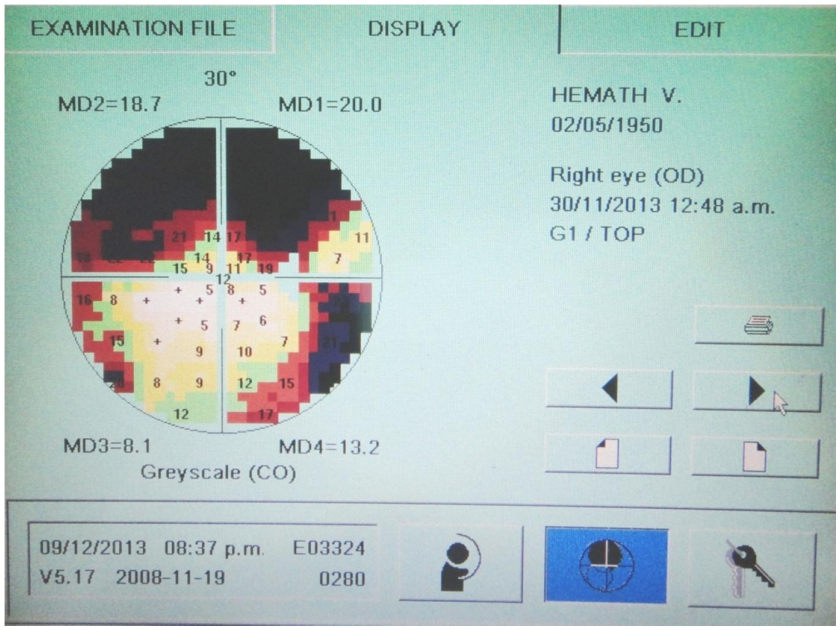


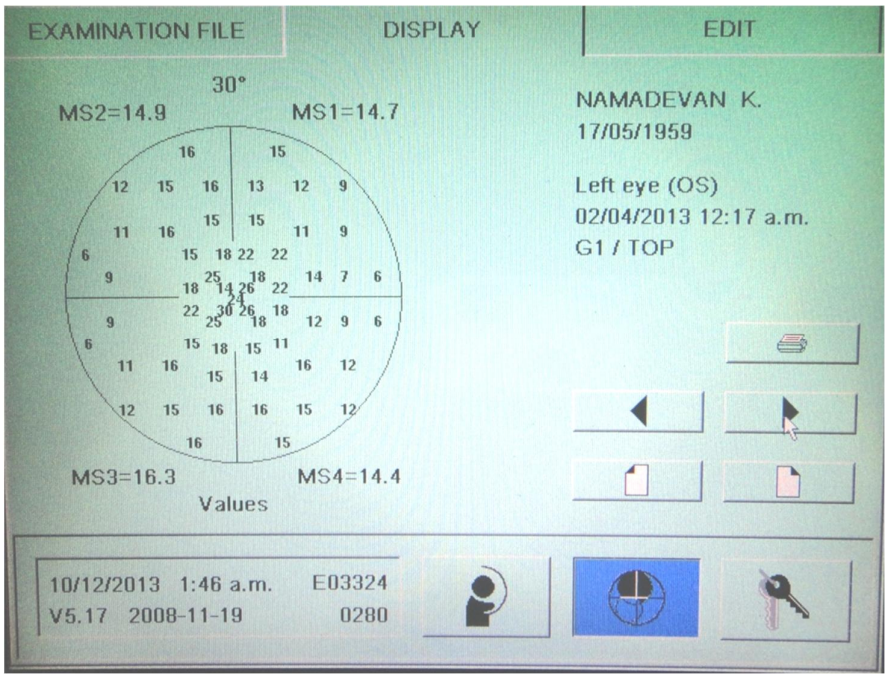


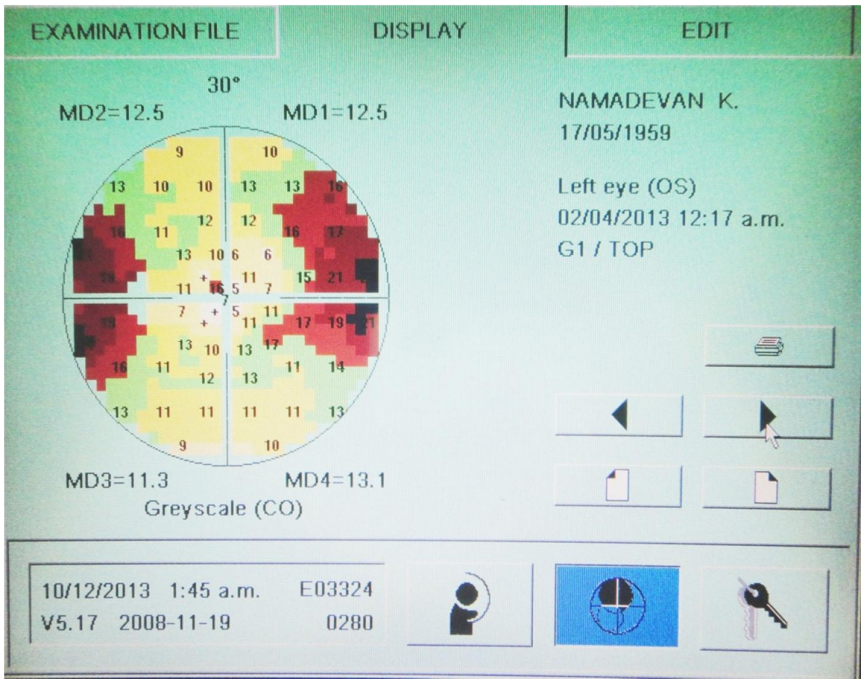
AUTOMATED PERIMETRY PICTURES





AUTOMATED PERIMETRY PICTURES





LIST OF ABBREVIATIONS

POAG	-	Primary Open Angle Glaucoma
HT	-	Hypertension
IOP	-	Intraocularpressure
TM	-	Trabecularmeshwork
ON	-	Optic Nerve
ONH	-	Optic Nerve Head
C/D RATIO	-	Cup Disc Ratio
NRR	-	Neuroretinal rim
OPP	-	Ocular perfusion pressure
BP	-	Blood Pressure
WHO	-	World Health Organisation
OR	-	Odds Ratio
D	-	Diopter

KEY TO MASTERCHART

M	-	Male
F	-	Female
RE	-	Right eye
LE	-	Left eye
BE	-	Both eyes
HT	-	Hypertension
CD	-	Cup disc
NRR	-	Neuroretinal rim
FBS	-	Fasting Blood Sugar
mgs	-	milligrams

SL.NO	NAME	AGE	SEX	BLOOD PRESSURE	DURATION OF HYPERTENSION	TREATMENT FOR HYPERTENSION	IOP	GONIO	FIELDS	FUNDUS	FBS mgs%
1	AKILA	75	F	140/90	10 YEARS	T.ATENOLOL 50mg 1-0-0 T.ENALAPRIL 2.5 mg 2-0-2	RE -20 LE -20	BE Grade-III	RE - NORMAL LE - SUPERIOR ARCUATE DEFECT	BE -Grade-II HT RETINOPATHY WITH RE - CD RATIO 0.6 LE - 0.6 ,INFERRIOR NRR NOTCHING	120
2	Thilakavathy	45	F	130/90	4 months	T.Amlodipine 2.5 mg 2-0-0	RE -20 LE -18	BE Grade-III	Normal	Normal	110
3	Rashiya	41	F	130/90	1 1/2 Years	T.Metoprolol 2.5 mg 0-0-1	RE -18 LE -16	BE Grade-IV	Normal	Normal	100
4	Esthar	70	F	150/90	8 Years	T.Enalapril 2.5 mg 2-0-2 T.Atenolol	RE -16 LE -14	BE Grade-III	Normal	Normal	102
5	Vinayagan	65	M	130/70	14 Years	T.Enalapril 2.5 mg 2-0-2 T.Atenolol 50 mg 1-0-0	RE -18 LE -18	BE Grade 0-1	Normal	CD ratio -0.6 Bayonetting sign + Laminar dot sign +	107
6	Chinnapan	45	M	140/90	5 Years	T.Amlodipine 2.5 mg 2-0-0	RE -20 LE -18	BE Grade-IV	Normal	Normal	88
7	Aadhilakshimi	55	F	140/80	4 Years	T.Amlodipine 2.5 mg 2-0-0	RE -20 LE -18	BE Grade-IV	Normal	Grade -I HT Retinopathy	84
8	Malliga	55	F	150/90	6 month	T.Enalapril 2.5 mg 2-0-2 T.Amlodipine 2.5 mg 2-0-0	RE -18 LE -16	BE Grade-III	Normal	Normal	94
9	Amla	60	F	150/80	10 Years	T.Amlodipine 2.5 mg 4-0-4 T.Enalapril 2.5 mg 4-0-4	RE -16 LE -16	BE Grade-IV	Normal	Grade -I HT Retinopathy	99
10	Swarnam	49	F	140/90	10 Years	T.Enalapril 2.5 mg 2-0-2 T.Amlodipine 2.5 mg 2-0-2 T.Atenolol 50 mg 1-0-0	RE -20 LE -18	BE Grade-III	Normal	Grade -I HT Retinopathy	104
11	Jayanthi	48	F	120/90	6 Months	T.Enalapril 2.gmg 1-0-1	RE -18 LE -18	BE Grade-IV	Normal	Normal	122
12	Ravi	56	M	136/90	8 Months	T.Enalapril 2.5 mg 4-0-4 T.Amlodipine 2.5 mg 4-0-1 T.Atenolol 50 mg 1-0-0	RE -20 LE -20	BE Grade-III	Normal	Grade-I HT Retinopathy	138
13	Malayappan	57	M	140/90	6 MonthS	T.Enalapril 2.5 mg 2-0-2 T.Amlodipine 2.5 mg 2-0-0	RE -20 LE -18	BE Grade-II	Normal	Normal	121
14	Rose	55	F	140/90	6 Months	T.Amlodipine 2.5 mg 2-0-0	RE -18 LE -16	BE Grade-III	Normal	Normal	126

15	Ezhilarasan	60	M	140/90	1 Year	T.Atenolol 50 mg 1-0-0 T.Enalapril	RE -16 LE -16	BE Grade-IV	Normal	Grade-I HT Retinopathy	83
16	**NAMADEVAN**	**54**	**M**	**150/90**	**4 YEARS**	**T.Amlodipine 2.5 mg 2-0-2 T.Enalapril 2.5 mg 1-0-0**	**RE - 24 LE - 26**	**BE Grade-III**	**RE- NORMAL LE- PARACENTRAL SCOTOMAS**	**RE - CD RATIO 0.6 BAYONETTING SIGN + LE - CD RATIO 0.7 BAYONETTING SIGN + NRR - CONCENTRIC THINNING +**	**86**
17	Chandran	54	M	160/110	5MonthS	T.Amlodipine 2.5 mg 2-0-0 T.Enalapril 2.5 mg 2-0-2	RE -20 LE -18	BE Grade-III	Normal	Grade-II HT Retinopathy	108
18	Selinmary	46	F	140/90	1 Year	T.Amlodipine 2.5 mg 2-0-0 T.Atenolol 50 mg 1-0-0	RE -16 LE -16	BE Grade II-III	Normal	Normal	106
19	Palkins	45	F	150/90	4 monthS	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 50 mg 1-0-0	RE -18 LE -14	BE Grade IV	Normal	Normal	120
20	Dhakshayini	43	F	150/90	7 monthS	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg 1-0-1	RE -14 LE -14	BE Grade II-III	Normal	Normal	120
21	Hemavathy	42	F	130/94	1 Year	T.Amlodipine 2.5 mg 2-0-0 T.Enalapril 50 mg 3-0-3	RE -18 LE -16	BE Grade III	Normal	Normal	126
22	Srinivasan	47	F	140/80	6 Month	T.Atenolol 50 mg 1-0-0	RE -20 LE -18	BE Grade III	Normal	Normal	131
23	RamaKrishnan	51	F	140/90	1 Year	T.Amlodipine 2.5 mg 2-0-0	RE -18 LE -16	BE Grade IV	Normal	Grade -I HT Retinopathy	98
24	Kuppu	61	F	120/80	1 1/2 Year	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg 1-0-1	RE -18 LE -16	BE Grade III	Normal	Grade -II HT Retinopathy	94
25	**SAROJA**	**65**	**F**	**130/90**	**10 YEARS**	**T.Enalapril 2.5 mg 2-0-2**	**RE -20 LE -18**	**BE Grade III**	**RE - CENTROCAECAL SCOTOMA LE - NORMAL**	**BE - GRADE II HT RETINOPATHY BE - CD RATIO O.7 LAMINAR DOT SIGN + BAYONETTING SIGN +**	96

26	Bavan	70	M	140/90	6 Months	T.Amlodipine 2.5 mg 2-0-2 T.Enalapril 2.5 mg 1-0-0	RE -20 LE -18	BE Grade IV	Normal	Normal	112
27	**KRISHNAN**	**61**	**M**	**140/90**	**6 YEARS**	**T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg 2-0-0**	**RE 28 LE 24**	**BE Grade III**	**RE - PARACENTRAL SCOTOMA LE - NORMAL**	**BE - CD RATIO 0.7 LAMINAR DOT SIGN + BAYONETTING SIGN + PERIPAPILLARY ATROPHY**	**108**
28	**DURGADEVI**	**55**	**F**	**130/90**	**15 YEARS**	**T.Amlodipine 2.5 mg 1-0-1**	**RE -22 LE -22**	**BE Grade IV**	**RE - SUPERIOR ARCUATE SCOTOMA LE - TUBULAR VISION**	**RE - CD RATIO 0.7 NRR - INFERIOR NOTCHING LE - CD RATIO 0.8 NRR - CONCENTRIC THINNING**	118
29	Mathiyalagan	53	M	140/90	7 Years	T.Enalapril 2.5 mg 2-0-2	RE -16 LE -16	BE Grade III	Normal	Normal	116
30	RamKumar	44	M	130/80	1 Year	T.Amlodipine 2.5 mg 1-0-0	RE -18 LE -18	BE Grade IV	Normal	CD Ratio o.5	84
31	Chitra	52	F	130/81	8 Years	T.Amlodipine 2.5 mg 1-0-0	RE -16 LE -14	BE Grade III	Normal	Normal	118
32	**VASUMATHY**	**52**	**F**	**140/90**	**15 YEARS**	**T.Enalapril 2.5 mg 1-0-1 T.Amlodipine 2.5 mg 1-0-0**	**RE -30 LE - 26**	**BE Grade IV**	**RE-DOUBLE ARCUATE SCOTOMA LE- SUPERIOR ARCUATE SCOTOMA**	**BE - GRADE II HT RETINOPATHY RE- CD RATIO 0.8 NRR - CONCENTRIC THINNING LE - CD RATIO 0.8 NRR - INFERIOR NOTCHING**	**116**
33	AbdulRahim	65	M	140/90	10 Years	T.Atenolol 50 mg 1/2-0-0 T.Metoprolol 2.5 mg 1-0-0	RE -18 LE -18	RE Grade II LE Grade III	Normal	Grade-II-Retinopathy	94
34	Santhanam	67	F	130/90	6 Months	T.Amlodipine 2.5 mg 2-0-0	RE -16 LE -16	BE Grade IV	Normal	Grade-II-Retinopathy	127
35	Devamathy	48	F	130/90	6 Years	T.Enalapril 2.5 mg 1-0-1 T.Amlodipine 2.5 mg 2-0-0	RE -16 LE -16	BE Grade III	Normal	Normal	124
36	Sasikala	54	F	140/90	6 Years	T.Amlodipine 2.5 mg 2-0-0	RE -16 LE -14	BE Grade IV	Normal	Normal	134
37	Jayakumar	47	M	140/91	6 Months	T.Enalapril 2.5 mg 1-0-1 T.Atenolol 50 mg 1-0-0	RE -18 LE -20	BE Grade II	Normal	Normal	128

38	Ramesh babu	63	M	140/92	5 Years	T.Amlodipine 2.5 mg 2-0-0 T.Enalapril 2.5 mg 2-0-2	RE -20 LE -20	BE Grade II	Normal	Normal	84
39	Bhooma	59	F	140/93	10 Years	T.Amlodipine 2.5 mg 2-0-0 T.Enalapril 2.5 mg 2-0-2	RE -18 LE -16	BE Grade III	Normal	Normal	96
40	Ramamoorthy	76	M	140/94	5 Years	T.Atenolol 50 mg 1-0-0 T.Enalapril 2.5 mg 2-0-2	RE -16 LE -14	BE Grade II	Normal	Grade II HT Retinopathy	92
41	SujathaDevi	60	F	140/90	10 Years	T.Amlodipine 2.5 mg 2-0-0 T.Enalapril 2.5 mg 2-0-2	RE -16 LE -16	BE Grade IV	Normal	Grade I HT Retinopathy	102
42	VijayaLakshmi	55	F	130/90	4 Years	T.Atenolol 50 mg 1-0-0	RE -16 LE -18	BE Grade III	Normal	Normal	118
43	Narayanamoorthy	64	M	130/90	3 Years	T.Amlodipine 2.5 mg 2-0-0 T.Enalapril 2.5 mg 1-0-1	RE -14 LE -16	BE Grade III	Normal	Normal	118
44	AnthonyKumar	70	F	130/80	5 Years	T.Enalapril 2.5 mg 1-0-1	RE -12 LE -12	BE Grade II	Normal	Grade II HT Retinopathy	128
45	**KHADHAR BHASHA**	**65**	**M**	**150/80**	**10 YEARS**	**T.Amlodipine 2.5 mg 2-0-0 T.Enalapril 2.5 mg 1-0-1**	**RE -20 LE - 20**	**BE Grade III**	**BE - PARACENTRAL SC**	**BE- GRADE II HT RETINOPATHY BE -CD RATIO 0.7 CONCENTRIC NRR THINNING BAYONETTING SIGN +**	**120**
46	Saroja	65	F	130/90	25 Years	T.Amlodipine 2.5 mg 2-0-0 T.Atenolol 50 mg 1-0-0	RE -16 LE -14	BE Grade II	Normal	Grade II HT Retinopathy	130
47	Anthoniammal	70	F	140/90	10 Years	Irregular Treatment	RE -15 LE -16	BE Grade III	Normal	Grade II HT Retinopathy	110
48	Kumar	60	M	130/90	5 Years	T.Amlodipine 2.5 mg 2-0-0 T.Atenolol 50 mg 1/2-0-1/2	RE -14 LE -14	RE Grade I LE Grade -II	Normal	Normal	104
49	Sudamani	45	F	130/90	2 Years	T.Amlodipine 2.5 mg 2-0-0	RE -16 LE -18	BE Grade III	Normal	Normal	88
50	UmaMaheswari	48	F	120/80	1 Year	T.Atenolol 50 mg 1-0-0	RE -14 LE -16	BE Grade IV	Normal	Normal	104
51	Bakiyam	68	F	140/90	7 Years	T.Amlodipine 2.5 mg 2-0-0 T.Enalapril	RE -12 LE -12	BE Grade III	Normal	Grade II HT Retinopathy	86
52	Shamala	50	F	120/80	2 Years	T.Atenolol 50 mg 1-0-0	RE -14 LE -14	BE Grade IV	Normal	Normal	113

53	ShahulHameed	50	F	130/90	6 Months	T.Amlodipine 2.5 mg 1-0-0	RE -16 LE -14	BE Grade IV	Normal	Grade I HT Retinopathy	94
54	Dinesh	43	M	130/80	1 year	T.Atenolol 50 mg 1-0-0	RE -18 LE -18	BE Grade III	Normal	Normal	108
55	Ravi	60	M	140/80	5 Years	T.Amlodipine 2.5 mg 2-0-0 T.Enalapril 2.5 mg 1-0-1	RE -18 LE -16	BE Grade II	Normal	Grade I HT Retinopathy	96
56	Usman	50	M	150/80	3 month	T.Atenolol 50 mg 1-0-0	RE -20 LE -20	BE Grade III	Normal	Normal	92
57	Uthaya	65	M	140/90	6 years	T.Enalapril 2.5 mg 1-0-1 T.Atenolol 50 mg 1-0-0	RE -20 LE -20	BE Grade IV	Normal	Grade II HT Retinopathy	108
58	Fathima	60	F	150/90	8 years	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg 2-0-2	RE -16 LE -16	BE Grade II	Normal	Normal	112
59	JayaKumar	70	M	140/80	10 years	T.Amlodipine 2.5 mg 2-0-2 T.Enalapril 2.5 mg 2-0-2	RE -14 LE -12	BE Grade III	Normal	Grade I HT Retinopathy	106
60	Devi	42	F	120/80	2 years	T.Atenolol 50 mg 1-0-0	RE -14 LE -14	BE Grade II	Normal	Normal	120
61	Indira	54	F	140/80	6 months	T.Atenolol 50 mg 1-0-0	RE -16 LE -16	BE Grade IV	Normal	Grade I HT Retinopathy	108
62	Ganesan	68	M	140/90	3 years	T.Enalapril 2.5 mg 2-0-2 T.Amlodipine 2.5 mg 2-0-0	RE -18 LE -18	BE Grade IV	Normal	Grade II HT Retinopathy	117
63	Narayanan	56	M	130/80	1 year	T.Amlodipine 2.5 mg 2-0-0	RE -14 LE -14	BE Grade II	Normal	Normal	116
64	Manjula	49	F	140/90	4 Years	T.Atenolol 50 mg 1-0-0	RE -14 LE -16	BE Grade IV	Normal	Normal	84
65	Selvarani	65	F	130/90	6 years	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg 1-0-1	RE -20 LE -20	BE Grade III	Normal	Grade I HT Retinopathy	94
66	Mohammed Ali	68	M	130/90	3 years	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg 1-0-1	RE -18 LE -18	BE Grade II	Normal	Grade II HT Retinopathy	108
67	Egavalli	46	F	130/80	3 mpnths	T.Atenolol 50 mg 1-0-0	RE -18 LE -16	BE Grade III	Normal	Normal	106
68	**JAMES**	**60**	**M**	**150/90**	**7 YEARS**	**T.Enalapril 2.5 mg 2-0-2**	**RE 24 LE 22**	**BE Grade IV**	**RE - INFERIOR ARCUATE DEFECT LE - NORMAL**	**RE - CD Ratio 0.7 NRR - SUPERIOR THINNING LE - CD RATIO 0.6**	**108**

69	Selvi	70	F	140/90	10 years	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg 1-0-1	RE -20 LE -20	BE Grade II	Normal	Grade III HT Retinopathy	116
70	Basha	44	M	140/80	3 years	T.Atenolol 50 mg 1-0-0	RE -18 LE -16	BE Grade III	Normal	Normal	127
71	Vasantha	68	F	140/90	3 years	T.Amlodipine 2.5 mg 2-0-2 T.Enalapril 2.5 mg 2-0-2	RE -18 LE -16	BE Grade III	Normal	Grade II HT Retinopathy	130
72	Manimaran	64	M	140/80	2 years	50 mg 1-0-0 T.Enalapril 2.5 mg 1-0-1	RE -18 LE -18	BE Grade IV	Normal	Grade II HT Retinopathy	123
73	Kalaivani	54	F	140/90	8 Months	T.Atenolol 50 mg 1-0-1	RE -16 LE -18	BE Grade IV	Normal	Normal	110
74	Amirtham	57	F	130/90	4 years	T.Enalapril 2.5 mg 2-0-2	RE -12 LE -12	BE Grade III	Normal	Normal	92
75	Ramya	59	M	150/90	6 years	T.Atenolol 50 mg 1-0-0 T.Enalapril 2.5 mg 1-0-1	RE -14 LE -16	BE Grade II	Normal	Normal	100
76	Lakshmi	48	F	140/80	5 months	T.Atenolol 50 mg 1-0-0	RE -16 LE -18	BE Grade II	Normal	Normal	109
77	Natarajan	55	M	130/90	6 years	T.Amlodipine 2.5 mg 2-0-2	RE -16 LE -16	BE Grade III	Normal	Normal	90
78	Jagatha	60	F	150/90	8 years	T.Amlodipine 2.5 mg 2-0-2 T.Enalapril 2.5 mg 2-0-2	RE -18 LE -16	BE Grade III	Normal	Normal	88
79	Shanmugam	56	M	140/90	1 year	T.Atenolol 50 mg 1-0-0	RE -18 LE -16	BE Grade IV	Normal	Normal	104
80	Meenakshi	58	F	130/90	1 year	T.Atenolol 50 mg 1-0-0	RE -16 LE -18	BE Grade IV	Normal	Normal	129
81	Thangaraj	62	M	130/80	5 Years	T.Atenolol 50 mg 1-0-0 T.Enalapril 2.5 mg 1-0-1	RE -18 LE -18	BE Grade IV	Normal	Normal	120
82	Nallamal	65	F	120/80	7 Years	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg 1-0-1	RE -20 LE -20	BE Grade III	Normal	Grade I HT Retinopathy	117
83	Rajamma	52	F	140/90	2 Years	T.Amlodipine 2.5 mg 2-0-2 T.Enalapril 2.5 mg 1-0-1	RE -20 LE -20	BE Grade III	Normal	Normal	96
84	Sarav	71	F	120/80	7 Years	T.Amlodipine 2.5 mg 2-0-2 T.Enalapril 2.5 mg 1-0-1	RE -16 LE -18	BE Grade III	Normal	Grade II HT Retinopathy	108

85	Thirumalai	60	M	120/80	2 Years	T.Atenolol 50 mg 1-0-0 T.Enalapril 2.5 mg 1-0-1	RE -14 LE -16	BE Grade II	Normal	Normal	134
86	**KANNIAH**	**79**	**M**	**158/90**	**5 YEARS**	**T.Amlodipine 2.5 mg 1-0-1** **T.Enalapril 2.5 mg 0-0-1**	**RE - 20** **LE - 20**	**BE Grade III**	**RE- INFERIOR ARCUATE DEFECT** **LE -SUPERIOR ARCUATE DEFECT**	**RE - CD RATIO 0.8** **NRR - SUPERIOR THINNING** **LE - CD RATIO 0.8** **NRR - INFERIOR THINNING**	**116**
87	Elumalai	56	M	140/90	8 Months	T.Atenolol 50 mg 1-0-0	RE -16 LE -16	BE Grade III	Normal	Normal	120
88	Anbalagan	53	M	140/90	1 Year	T.Atenolol 50 mg 1-0-0	RE -16 LE -16	BE Grade IV	Normal	Normal	110
89	Padmavathy	49	F	150/90	2 Years	T.Enalapril 2.5 mg 2-0-2	RE -14 LE -16	BE Grade III	Normal	Grade I HT Retinopathy	120
90	Deivanagi	64	F	140/90	2 Years	T.Amlodipine 2.5 mg 2-0-2	RE -14 LE -14	BE Grade IV	Normal	Normal	120
91	Thayappan	58	M	140/90	2 Years	T.Atenolol 50 mg 1-0-0	RE -14 LE -16	BE Grade IV	Normal	Normal	110
92	Loganathan	75	M	150/90	3 Years	T.Atenolol 50 mg 1-0-1 T.Enalapril 2.5 mg 1-0-1	RE -20 LE -20	BE Grade IV	Normal	Grade I HT Retinopathy	108
93	Karpagam	60	F	150/90	7 Years	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg 2-0-2	RE -16 LE -16	BE Grade III	Normal	Grade II HT Retinopathy	121
94	Sarala	78	F	160/80	10 Years	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg 2-0-2	RE -16 LE -14	BE Grade II	Normal	Grade III HT Retinopathy	86
95	Mariyapushoa	63	F	140/80	4 Years	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg -0-1	RE -18 LE -16	BE Grade III	Normal	Grade II HT Retinopathy	88
96	Devaraj	64	M	140/90	6 Years	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg -0-1	RE -20 LE -20	BE Grade III	Normal	Normal	104
97	Nagarathinam	69	M	140/90	7 Years	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg 0-0-1	RE -14 LE -16	BE Grade IV	Normal	Normal	94
98	Bavani	64	F	130/90	2 Years	T.Amlodipine 2.5 mg 1-0-1	RE -14 LE -16	BE Grade IV	Normal	Normal	128
99	RadhanKrishnan	66	M	130/90	5 Years	T.Amlodipine 2.5 mg 1-0-1 T.Enalapril 2.5 mg -0-1	RE -14 LE -14	BE Grade II	Normal	Grade I HT Retinopathy	100

100	Rajammal	53	F	120/80	5 Years	T.Atenolol 50 mg 1-0-0	RE -14 LE -12	BE Grade II	Normal	Normal	106

SL.NO.	NAME	AGE	SEX	B.P	IOP	GONIO	FIELDS	FUNDUS	FBS mgs%
1	Sathia moorthy	74	M	120/80	RE-20 LE-18	Grade III	Normal	Normal	120
2	Balan	43	M	110/70	RE-18 LE-16	Grade II	Normal	Normal	96
3	Dhanalakshmi	42	F	100/80	RE-14 LE-14	Grade III	Normal	Normal	110
4	Shanmuganathan	69	M	110/70	RE-14 LE-14	RE-Grade I LE-Grade II	Normal	Normal	110
5	Thangamuthu	65	M	120/80	RE-16 LE-16	Grade III	Normal	Normal	100
6	Ambika	44	F	110/70	RE-16 LE-18	Grade II	Normal	Normal	90
7	Muthukumar	54	M	120/80	RE-16 LE-14	Grade III	Normal	Normal	98
8	Elamathi	56	F	130/80	RE-16 LE-16	Grade IV	Normal	Normal	96
9	Noorjahan	57	F	120/80	RE-12 LE-12	Grade II	Normal	Normal	108
10	Moorthy	61	M	120/80	RE-16 LE-18	Grade IV	Normal	Normal	116
11	Arputham	48	F	110/70	RE-14 LE-14	Grade III	Normal	Normal	88
12	Mariambeevi	49	F	110/80	RE-18 LE-20	Grade II	Normal	Normal	98
13	Abdul Kahim	57	M	110/70	RE-14 LE-14	Grade IV	Normal	Normal	96
14	Neelavathy	61	F	110/70	RE-16 LE-18	Grade III	Normal	Normal	86
15	Jeevarathnam	56	M	120/80	RE-16 LE-16	Grade IV	Normal	Normal	102
16	Balu	54	M	130/70	RE-18 LE-16	Grade III	Normal	Normal	114
17	Eagambaram	61	M	120/80	RE-16 LE-16	Grade IV	Normal	Normal	128
18	Maheswari	53	F	130/70	RE-12 LE-12	Grade IV	Normal	Normal	130
19	Chinnaponnu	47	F	120/80	RE-12 LE-12	Grade III	Normal	Normal	118
20	Mariyamma	45	F	110/70	RE-16 LE-16	RE-Grade II LE-Grade III	Normal	Normal	136
21	Ashokan	42	M	110/70	RE-14 LE-12	Grade III	Normal	Normal	108
22	Mani	43	M	120/80	RE-14 LE-16	Grade IV	Normal	Normal	112

23	Sumathi	48	F	110/70	RE-14 LE-16	Grade III	Normal	Normal	116
24	Ramani	50	F	120/80	RE-18 LE-18	Grade III	Normal	Normal	98
25	**CHELLADURAI**	**62**	**M**	**110/70**	**RE-20 LE-20**	**Grade III**	**BE - SUPERIOR ARCUATE DEFECT**	**BE - CD RATIO - 0.6 INFERIOR NRR NOTCHING + BAYONETTING SIGN +**	96
26	**LOGANATHAN**	**65**	**M**	**110/70**	**RE-24 LE-22**	**Grade III**	**BE -NORMAL**	**RE - CD RATIO - 0.6 SUPERIOR NRR NOTCHING LE - CD RATIO O.5 BAYONETTING SIGN +**	128
27	RajaManikkam	44	M	120/80	RE-18 LE-16	Grade IV	Normal	Normal	126
28	Munusamy	70	M	130/80	RE-20 LE-18	Grade III	Normal	Normal	130
29	Valli	60	F	120/80	RE-18 LE-16	Grade IV	Normal	Normal	116
30	**YASODHA**	**71**	**F**	**110/70**	RE -24 LE -18	**Grade III**	**RE - SUPERIOR ARCUATE DEFECT. LE - GENERALISED DEPRESSION**	**RE - CD RATIO - 0.6 INFERIOR NRR NOTCHING + BAYONETTING SIGN + LE - CD RATIO O.5 BAYONETTING SIGN +**	**110**
31	Jayalatha	72	F	110/80	RE-16 LE-18	Grade III	Normal	Normal	99
32	Karuppaya	52	M	110/80	RE-14 LE-14	Grade III	Normal	Normal	108
33	Veeraragavan	45	M	120/70	RE-12 LE-12	Grade III	Normal	Normal	106
34	Seethama	54	F	120/70	RE-14 LE-14	Grade IV	Normal	Normal	116
35	Rahmathnisha	54	F	130/90	RE-16 LE-16	Grade III	Normal	Normal	120
36	Appavu	66	M	120/80	RE-14 LE-16	Grade III	Normal	Normal	122
37	Jahirhussain	68	M	110/70	RE-16 LE-18	Grade III	Normal	Normal	128
38	Malarvazhi	47	F	120/80	RE-14 LE-16	Grade IV	Normal	Normal	130
39	Radha	53	F	110/70	RE-14 LE-12	Grade III	Normal	Normal	104
40	Kamala	45	F	120/80	RE-16 LE-18	Grade IV	Normal	Normal	104

41	Eswariammal	62	F	110/70	RE-18 LE-18	Grade III	Normal	Normal	112
42	subramanian	56	M	110/70	RE-16 LE-14	Grade II	Normal	Normal	116
43	Vivekanandan	75	M	120/80	RE-16 LE-16	Grade III	Normal	Normal	117
44	Bharani	59	F	130/90	RE-18 LE-18	Grade III	Normal	Normal	180
45	Selvam	53	M	130/90	RE-14 LE-16	Grade IV	Normal	Normal	138
46	Dhanammal	72	F	120/90	RE-12 LE-12	Grade III	Normal	Normal	126
47	**MOHAMMED A**	**58**	**M**	**120/80**	**RE -26 LE -14**	**Grade III**	**BE - NORMAL**	**RE -CD RATIO 0.5 BAYONETTING SIGN + LE - CD RATIO 0.4**	**118**
48	Nagammal	67	F	110/80	RE-14 LE-16	RE -Grade I LE - Grade II	Normal	Normal	120
49	Parameshwari	52	F	110/70	RE-12 LE-14	Grade II	Normal	Normal	117
50	Pushparaj	51	M	110/60	RE-12 LE-12	Grade III	Normal	Normal	132
51	Malliga	49	F	110/70	RE-14 LE-14	Grade III	Normal	Normal	104
52	Mercy	51	F	120/80	RE-16 LE-16	Grade II	Normal	Normal	118
53	Palraj	66	M	110/60	RE-14 LE-16	Grade III	Normal	Normal	134
54	Ramarajan	61	M	120/60	RE-12 LE-12	Grade III	Normal	Normal	109
55	Krishnaveni	63	F	100/60	RE-12 LE-14	Grade II	Normal	Normal	98
56	Parvathy	69	F	120/80	RE-14 LE-16	Grade III	Normal	Normal	132
57	Rashiyabegam	48	F	110/70	RE-16 LE-14	Grade IV	Normal	Normal	96
58	Varadharajan	45	M	120/80	RE-16 LE-18	Grade IV	Normal	Normal	88
59	Dhanalakshmi	53	F	110/80	RE-16 LE-18	Grade IV	Normal	Normal	94
60	Kalavathy	69	F	130/90	RE-16 LE-16	Grade III	Normal	Normal	106
61	**HEMANTH**	**63**	**M**	**120/80**	**RE-30 LE -24**	**Grade IV**	**RE - SUPERIOR ARCUATE DEFECT. LE - PARACENTRAL SCOTOMA.**	**BE -CD RATIO 0.6 NRR THINNING + BAYONETTING SIGN + LAMINAR DOT SIGN +**	**114**
62	Asina	48	F	120/70	RE-16 LE-16	Grade III	Normal		88

63	Dhanammal	64	F	130/70	RE-16 LE-16	Grade III	Normal	Normal	86
64	chinnakannu	67	M	110/70	RE-16 LE-14	Grade III	Normal	Normal	94
65	Vijayalakshmi	45	F	120/80	RE-16 LE-14	Grade IV	Normal	Normal	120
66	Marimuthu	70	M	110/70	RE-18 LE-20	Grade IV	Normal	Normal	122
67	Anandhraj	43	M	120/80	RE-20 LE-20	Grade III	Normal	Normal	108
68	Mehrunisha	69	F	110/70	RE-14 LE-16	Grade II	Normal	Normal	106
69	Easwaran	68	M	110/60	RE-16 LE-18	Grade III	Normal	Normal	120
70	Aarumugam	53	M	120/60	RE-16 LE-14	Grade III	Normal	Normal	88
71	Chellamal	56	F	130/70	RE-16 LE-14	Grade III	Normal	Normal	94
72	kantha	55	F	120/80	RE-14 LE-14	Grade IV	Normal	Normal	120
73	PremKumar	47	M	110/80	RE-16 LE-16	Grade IV	Normal	Normal	82
74	Durairaj	59	M	100/70	RE-12 LE-14	Grade III	Normal	Normal	89
75	Rosemary	60	F	110/60	RE-16 LE-16	Grade III	Normal	Normal	109
76	SivaKumar	55	M	110/70	RE-14 LE-12	Grade III	Normal	Normal	118
77	Shanthi	57	F	120/80	RE-16 LE-16	Grade II	Normal	Normal	114
78	Ramanan	56	M	130/80	RE-16 LE-14	Grade IV	Normal	Normal	86
79	Sameerabegum	53	F	140/80	RE-20 LE-18	Grade III	Normal	Normal	94
80	Lakshmi	63	F	130/70	RE-18 LE-18	Grade II	Normal	Normal	92
81	Neelavathy	64	F	120/80	RE-18 LE-18	Grade III	Normal	Normal	132
82	Jeevarathnam	70	M	110/70	RE-20 LE-20	Grade III	Normal	Normal	138
83	**MOHANAMBAL**	**57**	**F**	**120/80**	**RE -26 LE -20**	**Grade III**	**RE - INFERIOR ARCUATE DEFECT LE - NORMAL**	**RE -CD RATIO 0.6.NRR-SUPERIORNOTCHING. LE - CD RATIO 0.5**	**121**
84	Malathi	68	F	110/70	RE-14 LE-14	Grade II	Normal	Normal	127
85	Abdul Rahim	54	M	120/70	RE-14 LE-16	Grade II	Normal	Normal	83

86	UmaDevi	47	F	120/80	RE-12 LE-14	Grade II	Normal	Normal	94
87	Palaniammal	63	F	110/80	RE-14 LE-16	Grade III	Normal	Normal	89
88	Singaram	57	M	100/60	RE-16 LE-14	Grade III	Normal	Normal	136
89	Shanthamani	74	F	120/80	RE-20 LE-20	Grade III	Normal	Normal	118
90	Muhunthan	51	M	110/70	RE-16 LE-16	Grade II	Normal	Normal	94
91	Gandhimathi	77	F	120/80	RE-16 LE-18	Grade III	Normal	Normal	98
92	Subhaiah	62	M	110/70	RE-14 LE-14	Grade IV	Normal	Normal	108
93	Renugamhal	63	F	120/60	RE-12 LE-12	Grade III	Normal	Normal	102
94	Sorooharani	67	F	100/60	RE-14 LE-14	Grade II	Normal	Normal	88
95	Ayyappan	62	M	110/80	RE-12 LE-14	Grade IV	Normal	Normal	94
96	Gowri	47	F	120/80	RE-14 LE-14	Grade IV	Normal	Normal	96
97	NahishaBanu	65	F	130/70	RE-18 LE-18	Grade III	Normal	Normal	121
98	Manikkam	52	M	120/80	RE-20 LE-18	Grade II	Normal	Normal	128
99	Kalaivani	63	F	120/80	RE-18 LE-16	Grade III	Normal	Normal	120
100	Usharani	62	F	110/70	RE-16 LE-14	Grade IV	Normal	Normal	98